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Harry Rainy M.A., F.R.C.P.Ed., F.R.S.E.

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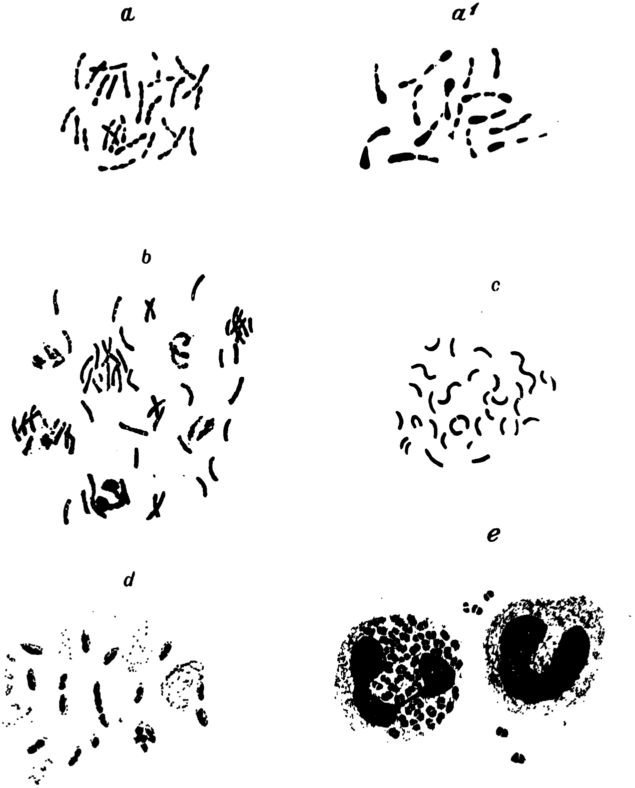

BACTERIA (pp. 575–591).

(*After an Original Drawing by Richard Muir.*)

a, Bacillus diphtheriæ. Long forms. 24 hours' growth; agar cult; staine
watery methyl blue; × 1,000.

a', The same after 5 days' growth. Involuted forms.

b, Bacillus tuberculosis in sputum, from case of phthisis. Stained Z. Neelsen
× 1,000.

c, Spirillum choleræ. 24 hours' growth; agar cult; stained fuchsine; × 1,000.

d, Pneumococcus (Fraenkel's) in sputum, from case of acute pneumonia. Staine
Z. Neelsen, fuchsine; decolorised in weak acetic acid; × 1,000.

e, Gonococci in gonorrhœal pus. Stained thionine blue; × 1,000.

[***Frontispiece.***

A Kampmeier
11 - 1904

CLINICAL METHODS

A GUIDE TO THE PRACTICAL STUDY OF MEDICINE

BY

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WITH UPWARDS OF 150 ILLUSTRATIONS AND 8 COLOURED PLATES

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PREFACE TO THE ORIGINAL EDITION.

THE title "Clinical Methods" probably describes the scope of this book better than any other. It is not intended as a treatise upon medical diagnosis. On that subject there is already a sufficiency of good works in existence. It aims rather at describing those methods of clinical investigation by the proper application of which a correct diagnosis can alone be arrived at. To every student when he first begins work in a medical ward the question presents itself: How shall I investigate this case? To that question the present work is intended to provide an answer. The first chapter deals, therefore, with the methods of case-taking in general, and includes a general scheme for the investigation of medical cases. The rest of the book is really an expansion of that scheme, each system being taken up separately, and the methods of investigating it described in detail.

A special chapter has been devoted to the clinical methods of examining children, as these differ in many respects from those employed in the case of adults. Chapters have also been added on the examination of Pathological Fluids and on Clinical Bacteriology, subjects which are daily growing in importance. The methods employed in the investigation of surgical, gynæcological, or obstetric cases do not fall within the scope of the work.

No effort has been spared to make the book thoroughly up to date, and it is hoped, therefore, that it will be found useful by those practitioners who may wish to make themselves acquainted with the latest methods of clinical investigation. While the whole

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book has passed through the hands of both of us, yet each has made himself specially responsible for certain parts. Thus Dr. Rainy has written Chapters II., IV., VI., and XIV., the sections on the electrical examination of muscles and nerves, on the parasites of the alimentary tract, and on the microscopical examination of the urine. The rest of the work is from the pen of Dr. Hutchison.

In order to avoid burdening the text, but few references have been given to authorities and original sources. We should like, however, to take this opportunity of acknowledging the help which we have received from various friends. Amongst these are Drs. Alex. Bruce, R. W. Philip, G. Lovell Gulland, and John Thomson, who have helped us with criticism and advice in the preparation of Chapters IV. and IX., VI., V., and XII. respectively. We have also to thank Dr. Patrick Manson, Dr. Byrom Bramwell, Dr. J. Purves Stewart, and Prof. Symington for the use of specimens and illustrations, and Dr. T. F. Milroy for assistance in the revision of proofs. To Dr. R. J. M. Buchanan we are specially indebted for preparing the drawings illustrating the microscopical examination of the blood.

R. H.
H. R.

September, 1897.

PREFACE TO THE SECOND EDITION.

WHILST the general plan of this manual remains unchanged, we have taken advantage of the call for a new edition to revise it thoroughly, and, as far as possible, to bring it up to date. Several new figures have also been introduced.

Many methods which have been recently proposed are not included in the volume; some because they have not yet been sufficiently proved; others—and this holds true especially of chemical analyses—because they are too complicated for clinical use where simpler, though less accurate, procedures suffice.

The chief alterations of this edition will be found in Chapters V., IX., and XIV., of which many parts have been entirely rewritten; but in almost every chapter some changes have been made. The index has also been considerably improved by the kind assistance of Mr. Hewitt.

In addition to those whose help has been already acknowledged, we desire to thank Professor Wyllie and Drs. Edwin Bramwell, Henry Head, and Stuart McDonald for much valuable advice during our work of revision.

R. H.
H. R.

March, 1902.

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CLINICAL METHODS.

CHAPTER I.

CASE-TAKING.

THERE can be no question of the value of accurate and systematic case-taking. It trains the beginner in habits of thoroughness and exactness at the bedside, and ensures that no point of importance in the case is missed. To the more experienced clinician the systematic record of cases is of no less value. It gives to his experience a concrete embodiment, so that he can draw upon it at any future time by the comparison of new cases with old, and so enables him gradually to build up his clinical knowledge upon a sure foundation. When we come to the *method* to be pursued in taking a case, however, it is a question of "*quot homines, tot sententiæ.*" Almost every clinical teacher has his own particular plan for investigating and recording a case. Nor is it of so much importance what particular method one adopts, provided he adheres to it. Every good method of case-taking should be both comprehensive and concise. It should be comprehensive, so as to be capable of being applied to every case and of covering all the points in it; it should be concise, so as to present all the important features of a case in as small a compass as possible. The question of conciseness is of very great importance. Nothing is more annoying than to be obliged to wade through a mass of verbiage in order to get at the chief facts of a particular case.

The student should practise the art of focussing a case in such a way as to present its leading features in a few sentences. For this object the writing of *résumés* of cases will be found a useful exercise. He should also avoid lengthy verbal descriptions as far as possible, especially where the facts admit of graphic representation. The outline diagrams now supplied by Messrs. Lewis, Danielsson, and others will be found useful aids in this respect. Physical signs can be filled in on them by means of conventional symbols.

We have appended to this chapter a scheme of case-taking which meets all necessary requirements. At the same time, it must be used, like all such schemes, with some judgment and elasticity. All the points mentioned need not be minutely inquired into in each individual case. For example, if a patient is suffering from advanced cardiac disease, there is no use in writing a minute description of the state of his teeth. Yet that is the kind of error into which beginners not unfrequently fall. Of course, it demands some experience to enable one to say what *are* the points which it is of importance to inquire into in any particular case, and at first one may sometimes be at fault; but the application of a little commonsense will ensure the avoidance of any gross blunders.

The "taking" of any case consists of two parts—

I. **The interrogation of the patient.**

II. **The physical examination.**

I. The object of the **interrogation of the patient** is to elicit information regarding his present illness and the state of his previous health and that of his family. The interrogation must be pursued with patience, the patient being allowed, as far as possible, to tell his story in his own words. Two good rules should be remembered—first, to avoid leading questions; and, secondly, never to ask the same question twice. The use of leading questions is only occasionally allowable, as, for instance, when one suspects that he has to do with a case of malingering, when one may get the patient to assert the existence of contradictory symptoms, so confirming one's suspicions. It may also be necessary in dealing with patients who are stupid either by nature or as the result of disease. When one is trying to elicit what are known as "subjective symptoms," the use of leading questions may also be admissible. It is important to avoid asking the same question twice, because to do so looks careless, and conveys to the patient the impression of taking but a languid interest in his case.*

We may proceed now to go more into detail regarding the questions which should be asked. In doing so, we shall consider first the questions which one has to put in every case—what one may call the *general interrogation*—and then we shall take up the questions which have to be put in examining cases of disease affecting the different systems or organs; this may be called the *special interrogation.*

1. **General interrogation.**—Begin by ascertaining the patient's name, age, occupation, and whether he is married or single. It is also of importance to note his exact postal address for purposes of future communication.

Two important questions then follow—(1) Of what does he complain? † (2) How long have the symptoms been present? Having thus defined his complaint and its duration, proceed to ascertain the chief facts in his history.

---

* When one has to deal with a patient who is very deaf it will often be found convenient to use a binaural stethoscope as a speaking trumpet, the ear pieces being placed in the patient's ears while one speaks into the chest piece.

† It is a mistake to ask "What is the matter?" as this lays one open to the retort that that is what the patient came to find out.

The most logical plan is to take the *family history* first.* It is usually sufficient to inquire regarding the state of health or cause of death of the immediate relatives only—the parents, brothers and sisters, and, if the patient be married, of his own children, if he has any. These facts tell us whether he is predisposed by heredity to any particular disease.

One may then pass to his *personal history*. Here it is well to begin with what may be grouped together as the patient's environment, or *surroundings* and *habits*. This includes inquiry into (*a*) the exact nature of his occupation, and whether or not it exposes him to injurious influences; (*b*) his home surroundings, their sanitary condition or otherwise; (*c*) the amount of exercise he takes; (*d*) the nature of his food, and the amount of his indulgence in such articles as alcohol, tea, and tobacco.† Regarding alcohol, it is important to ascertain not merely how much—*e.g.* how many glasses of spirits per day—the patient takes, but how and when he consumes it—*e.g.* with or between meals. The kind of tobacco he smokes and the number of ounces consumed per week may require to be ascertained in some cases. (*e*) One should always ascertain, lastly, whether or not he has ever lived abroad, and, if so, in what part of the world.

The information thus acquired enables one to add to the tendencies to disease which the patient has inherited, those which he has acquired from his environment and personal habits.

One should take up next the question of the patient's *previous health.* Ascertain what illnesses he has had, when he had them, their duration, and whether or not his recovery from them was complete. It is usually necessary to inquire directly as to whether or not the patient has ever had syphilis. It is not sufficient to ascertain that he has had a sore; the question of secondary symptoms—*e.g.* rash—must also be gone into. If the patient denies syphilis, it may be necessary to ask whether or not he has ever been exposed to the risk of it, and whether or not he has had any other venereal disease. In the case of female patients, information regarding venereal disease should, as far as possible, be obtained indirectly, direct interrogation on the subject being employed only when a definite history is absolutely necessary for the elucidation of the case. The student need hardly be reminded that even under these circumstances his questions should be put as delicately as possible.

---

* This statement is perfectly true in theory, but in practice it is perhaps more convenient to begin with the history of the present illness, to pass from that to an inquiry into the patient's previous health, and from thence to the family history.

† When inquiring into a patient's habits of life it will often be found advantageous, particularly in private practice, to ask him to give a brief account of a typical day.

Having now ascertained the patient's inherited and acquired tendencies, and the seeds of disease which may have been sown in him by his previous illnesses, we are ready to acquire information regarding his present disorder.

Ask how and when it began, whether suddenly or gradually; what was the first thing he noticed wrong; what has been the order of appearance of his symptoms, and which are those that chiefly trouble him at the present time. Ascertain whether or not he has already been under treatment, and, if so, what has been done for him.

This exhausts the general interrogation, and includes the chief facts that have to be inquired into in every case.

2. The **special interrogation**, to which we have already referred, must be modified according to the particular organ which is believed to be affected and

the nature of the disease of which it is suspected to be the seat. It is here that the student has most difficulty. It is only by experience that one can tell what it is essential to ask in each individual case. In order to help the beginner, however, we have drawn up for his guidance a scheme of interrogation which he can pursue when he has reason to suppose that the patient's general symptoms point to an affection of any particular system or organ. Such a scheme is necessarily very far from complete, and may require to be supplemented in individual cases. Nor is one able in such a work as this to explain *why* such and such questions should be put in affections of this or that organ or system. The reasons for the questions the student will find out for himself in due time. Our present object is merely to help him in the interrogation of his earlier cases, so that he may not miss any important facts. The questions are to a considerable extent concerned with eliciting what are sometimes spoken of as "*subjective symptoms*"—*i.e.* the morbid sensations experienced by a patient as the result of the disease of some organ or system.

In making the notes, these, along with the other replies, should be entered under the special system to which they refer.

1. Alimentary system and abdomen.*

(*a*) Symptoms point to an affection of the stomach. Inquire regarding—

Appetite.—Is it excessive, diminished, or capricious? Does it increase on eating? Does he suffer from thirst?

Meals.—Arrangement of these; the nature of the food. Does he eat between meals?

---

* While the methods of gynecological examination are beyond the scope of this book, yet it must not be forgotten that it is frequently necessary in purely medical cases to inquire regarding the menstrual function. The cases in which such inquiry may be required are too diverse to be enumerated here. It need hardly be said that the necessary questions should not be abruptly put

Sensations referred to stomach.—Their nature, and where exactly they are felt. Their relation to the taking of food; are they produced or relieved by it? How long after food do they come on? Are they specially influenced by different kinds of food?

Vomiting.—Frequency and time of; by day or by night; in the morning or in the evening. Its relation to food; is it only after food, or does it occur at other times? Its relation to pain; does it relieve pain or not? Does patient strain and retch much, or does the vomited matter come up quite easily?

General characters of vomited matter.—Its amount and colour. Is there ever "coffee-grounds" vomiting; is it ever sour and frothy?

Eructations.—Presence or absence; have they any taste?

Flatulence.—Presence or absence; after food only or between meals? Relation to particular articles of food. Does it tend to escape downwards or upwards?

State of the bowels.—How often are they opened? Any special characters of the motions.

(*b*) Symptoms point to an affection of the intestines. Inquire regarding—

Diarrhœa.—Its frequency and its relation to meals or to special articles of food. Character of the motions. Has he ever passed any blood or slime? Is there any straining or tenesmus during defæcation? Is there any flatulence?

Constipation.—What is his usual habit? are the bowels opened regularly, and if so, how often? How long since the last motion? Has he ever noticed any grooving or flattening

---

to the patient, but should be delicately led up to. Having inquired regarding the regularity of the bowels, one may ask if the patient is "regular in her own health," or "regular in her unwell times." It will be remembered that in a majority of cases menstruation recurs every twenty-eight days, but the intervals may be longer or shorter according to the patient's habit. If menstruation has ceased, one must inquire how long it has been absent. Normally the cessation of menstruation, or menopause, should not occur till about the forty-fifth year. It is also necessary to inquire whether the patient is losing more or less blood than usual. This is specially indicated in cases of anæmia. If the menstrual flow lasts for less than two or more than eight days, it is to be regarded as abnormal. The presence or absence of pain at the period is also a point of some importance. The age at which menstruation began, and the occurrence or not of intermenstrual leucorrhœa ("white discharge") must sometimes be inquired into.

of the motions? Does the constipation alternate with diarrhoea? Has he any griping pain? Has he had any vomiting?

Pain.—Character; persistent or intermittent. Where is it felt worst? Is it relieved or aggravated by pressure?

(*c*) Symptoms point to an affection of the liver —*e.g.* patient is jaundiced, or has pain in region of liver. Inquire regarding—

Pain.—Its site. Has he ever any attacks of very severe pain, coming on suddenly and lasting for a few hours? If so, did the pain radiate, and in what direction? Was there vomiting with it? Was he yellow at all after it subsided? Has he ever pain in the tip of the shoulder?

Does he suffer from piles?

Does he ever vomit blood?

Has he noticed any change in the colour of the urine or fæces?

Does his skin itch at all (if he is jaundiced)? Inquire also regarding his digestion on the lines of the interrogation already laid down for affections of the stomach.

2. The symptoms point to an affection of the **circulatory system.**

Inquire regarding—

A *family history* of gout, rheumatism, angina, apoplexy, or heart disease.

A personal history of rheumatic fever, St. Vitus' dance, scarlatina, or diphtheria. (If a child, ask also about sore throats and "growing pains.")

The following subjective sensations:—

Dyspnœa.—Has he to sit up in bed, or can he sleep lying down? When does it come on? *Præcordial pain* or distress; its exact site and character; does it radiate or not? If so, in what direction? *Palpitation:* its relation to meals, and to exertion. Does the heart give an occasional thump now and then? *Sleep,* good or bad; does he dream? *Giddiness,* is it ever present, and when?

Ask also for signs indicative of general venous distension —*e.g.* do the feet ever swell? Has he any cough? What is the state of the digestion? Does his nose ever bleed?

3. The symptoms and appearances point to an affection of the **blood.** Inquire regarding—

Family history of bleeders. Has he had any loss of blood?

Has he bleeding piles? (If a woman—is menstruation excessive or diminished?) What is the state of the bowels?

Any possibility of lead poisoning or malaria?

Such subjective sensations as breathlessness on exertion; headache; giddiness.

Do the feet ever swell?

4. The symptoms point to an affection of the **respiratory organs.** Inquire regarding—

Family history of bronchitis, asthma, phthisis, or "scrofula." The patient's occupation; does it expose him to the inhalation of irritating fumes or particles? Has he ever had large glands in the neck? Does he sweat at night? Is he getting thinner?

Cough.—Its character and frequency; when is it worst? Does it pain him or not? Does he ever vomit with it?

Expectoration.—Its amount and general characters; yellow or not? Ever blood in it? If so, is it only after severe coughing? Is the blood bright and frothy or dark in colour?

Pain in chest.—Is it aggravated by taking a breath? Constant or not? Where seated?

Dyspnœa.—When is it felt? If spasmodic, ask him to describe an attack.

5. The symptoms point to an affection of the **Kidneys**—*e.g.* general dropsy—or **urinary passages**—*e.g.* pain in micturition. Inquire regarding—

Family history of Bright's disease, gout, or apoplexy.

Personal history of scarlatina, syphilis, lead-poisoning, prolonged suppurations, gravel or gout, and previous renal disease.

Has he any pain in the lumbar region? Ever any attacks of acute pain shooting down into the groin?

The following remote symptoms:—

Headache, vomiting, drowsiness, paralysis or fits, dimness of sight, dyspnœa.

Does the face ever look puffy in the morning?

What is the state of the bowels?

Inquire regarding micturition as follows:—

Urine.—Is it altered in amount? Has he to rise in the night to pass it?

Is it altered in colour? Is it clear or turbid when passed? Ever any blood in it? If so, at what period of micturition is it present?

Is there any increased frequency of micturition? Is the increase by day or by night?

Is there any pain during micturition? Is it before, during, or after the act? What is its character, and where is it felt? Is it aggravated by movement?

6. In **skin diseases.**

Inquire carefully into the patient's personal habits as regards diet, clothing, and washing. Ask if he has been taking any drugs recently. It may be necessary to inquire carefully regarding syphilis. Does the eruption itch? If so, when is the itching worst? Did the eruption appear all at once or in crops? (Family history of gout; previous history of rheumatism, anæmia, etc.)

7. The symptoms point to an affection of the **nervous system.**

Inquire regarding—

A *family* history of mental disease, St. Vitus' dance, paralysis, or fits.

The nature of the patient's work; is he exposed to any poisons—*e.g.* lead, mercury, arsenic, naphtha, etc. Syphilis and alcohol should be inquired about with special care.

In cerebral cases it is often very important to inquire regarding discharge from the ear.

Should he complain of *fits*, the following questions should be asked:—

Age at first fit? Any assigned cause? Describe the first fit. When did the second occur? What has been shortest and longest interval between the fits? Are they more or less frequent now? Do they occur in sleep or not? Has he any premonition or aura? What is its character? How long before the loss of consciousness does it occur? Is the onset sudden or gradual? Are convulsions present? Are they general or local? Where do they begin and end? Does he fall? Has he ever hurt himself? Does he bite his tongue, micturate, or defæcate during the fit? Are there any after-symptoms, such as sleep, headache, automatism, or paralysis? Is there any subsequent mental disturbance?

If he complains of *paralysis,* inquire regarding—

Symptoms of heart disease, or chronic renal disease (*see* Circulatory and Urinary Systems). Had he any premonitory symptoms before the onset? Has he any headache or vomiting? Where is the headache situated? Has he any giddiness or difficulty in walking? (The method of eliciting other subjective symptoms of nervous disease is considered along with the investigation of the cranial nerves.)

8. The symptoms point to an affection of the **bones** or **joints.**

Inquire specially, in the family history, for tubercular disease, rheumatism, gout, or syphilis, and in the personal history for tubercular disease, previous manifestations of gout or rheumatism, for syphilis or gonorrhœa, and for any remote or recent injury (and in a woman for leucorrhœa or post-partum trouble).

If there be pain referred to a bone, ask whether it is worse during the day or during the night. If the pain be in a joint, ask whether it is constantly present or only when the joint is moved. Are there any starting pains at night? Is the pain affected by weather? Does the pain shift from one joint to another?

If the patient is a young **child,** the following special questions should be put to the mother* :—

How many other children are there? Any dead, and of what? Where does patient come in the family? Have there been any miscarriages? If so, when? Health of father's and mother's family? Mother's health during pregnancy?

Was this a full-time child? Was the labour normal? Was the child breast-fed: if so, how long? If not, how was it fed? What food does it get now? Had it any rash after birth, or any snuffles? When did it begin to get its teeth and to walk?

What is the usual state of the digestion and bowels?

Inquire regarding previous illnesses :—Fits (number and dates), attacks of diarrhœa, vomiting, sore-throat or bronchitis. Infectious diseases and ages at which they occurred (measles, whooping cough, chicken-pox, scarlatina, etc.). Has there been any running from the ears? If the child has a cough inquire whether it has ever whooped, when the attacks are worst, and whether the cough is ever followed by vomiting.

The interrogation of the patient being completed, one proceeds to—

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* In the absence of the mother, note from whom the history was obtained.

II. **The physical examination.**

One investigates first of all, in every case, the patient's general state. This includes the general condition of his nutrition, the presence of any obviously morbid appearances, and the other points considered in detail in Chapter II. One proceeds after that to the investigation of each system by itself. What system should be taken up first? As regards this, there are two possible methods. One may either take up the systems in one and the same order in every case, beginning, say, with the Alimentary, or one may examine first the system which is most affected. The latter is, on the whole, the better plan, provided always that one is able to tell which system it really is that is most diseased. The advantage of this method is that it gives most prominence to the most important part of the physical examination. Whichever plan the student elects to adopt, he may now proceed to the physical examination of the different systems in accordance with the instructions laid down in the following chapters, the results being noted in the order given in the scheme below.

Only one more point regarding case-taking remains to be emphasised, and that is the importance of noting negative as well as positive facts. It is often quite as essential, for example, to state that such a symptom as dyspnœa is absent as to record the fact of its presence. This is a point the importance of which is apt not to be fully appreciated by the beginner.

In conclusion, it need hardly be said that the examination should be carried out as gently as possible, all unnecessary exposure, exhaustion, or chilling of the patient being carefully avoided. If the patient is suffering from severe or acute disease, it may be advisable to postpone all physical examination other than that which is absolutely necessary to the diagnosis of his condition, or for guidance in treatment. It should also be borne in mind that when a patient is much exhausted, or suffering from serious disease of the lungs or heart, very dangerous and even fatal results may ensue if he be thoughtlessly made to sit up in bed in order to have his chest examined.

CASE-TAKING SCHEME.

I. INTERROGATION.

Name. Age. Occupation. Married or single. Address. Date of coming under observation.

Complaint.

Duration.

Family history.*—Inquire regarding parents, brothers and sisters, and patient's own children. Note state of their health, or the cause of their death, with age at which they died.

Personal history.

Environment.—Nature of work and its surroundings. Hygienic conditions at home; habits as to exercise, food, tea, alcohol, and tobacco.

Previous illnesses or accidents (if any), with their time of occurrence, duration, and results.

Present illness.—Time and mode of its origin, the order in which symptoms appeared, and the chief symptoms which trouble patient now; treatment (if any) already employed.

II. PHYSICAL EXAMINATION.

1. Present state.

General condition.—General state of consciousness and intelligence. Decubitus (if in bed), or attitude and gait (if up) (p. 19). General state of development and nutrition. Expression of face; presence or absence of pallor, jaundice, cyanosis, dropsy, or trophic changes. Presence or absence of any special characters of the hands (p. 32). Glandular enlargements. Character of the respiration, and the presence or absence of cough. Take the temperature.

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* *See* footnote, p. 4.

2. **Alimentary system.**

Subjective symptoms (*see* special interrogation).

Examine the *mouth* (including the teeth, gums, and tongue), the *pharynx*, and *fauces* (pp. 46–50), and the *œsophagus* (with use of sound if necessary) (p. 51).

General inspection, palpation and percussion of the *abdomen* (pp. 54–64).

Stomach.—Palpation and percussion (pp. 64–69). Examination of gastric contents (test breakfast) or vomit.

Intestines.—Investigation of (p. 81). Rectal examination if necessary (p. 82). Examination of fæces (p. 93).

Liver and Gall Bladder.—Examination of by palpation and percussion (pp. 69–75).

Spleen.—Examination of (p. 75).

3. **Circulatory system.**

Heart.—Subjective symptoms (*see* special interrogation).

Pulse.—Describe its rate and its rhythm. Compare the force of successive beats. Ascertain the state of the vessel walls. Note the blood pressure during and between the beats. Observe the amplitude of the pulse waves. Analyse a complete beat of the pulse regarding rise, maintenance, and fall of pressure, and determine the presence or absence of secondary waves. Take tracings if the pulse is abnormal.

Inspection and palpation of *præcordia*, noting position and character of apex beat, presence or absence of epigastric pulsation or præcordial thrills, or of pulsation in the neck or at the base of heart.

Percussion of *Heart* (p. 129).

(*a*) Upper border }
(*b*) Right border } superficial, deep.
(*c*) Left border }

Auscultation of *Heart* (p. 145).

(*a*) At apex and a little internal to it.
(*b*) Tricuspid area at lower end of sternum.
(*c*) Aortic area.
(*d*) Pulmonary area and a little outside it.
(*e*) Between base and apex (third and fourth left costal cartilages).
(*f*) Veins and arteries of neck.

If a bruit is heard, note :—

(*a*) Its time.
(*b*) Its character (musical, harsh, etc.).
(*c*) Its point of maximum intensity.
(*d*) Its direction of propagation.

4. The blood.

Count the red and white corpuscles (pp. 200–206). Estimate the hæmoglobin (pp. 209–216). Examine the blood microscopically, making films if necessary (pp. 216–230).

5. Respiratory system.

Subjective symptoms (*see* special interrogation).

Count the respirations and describe their character.

Inspection of chest, noting its shape, power of expansion, etc. (p. 239).

Mensuration of the two sides of the chest.

Palpation of chest (expansion and vocal fremitus) (p. 249).

Percussion of lungs anteriorly, laterally, and posteriorly (p. 261).

Auscultation of lungs in same order (p. 266), noting—

(*a*) Type of breath sounds.

(*b*) Character of vocal resonance.

(*c*) Presence or absence of accompaniments.

Sputum.—Note its naked eye and microscopic characters (p. 284).

6. Urinary system.

Palpate the *Kidneys* (p. 78).

Examine the *Urine.* — Physically (p. 295), chemically (p. 307), microscopically (p. 364), making a note in *every* case of the following points :—

Quantity in twenty-four hours, colour, specific gravity, reaction, odour, general character of deposit.

Presence or absence of albumin, blood, sugar, and bile.

Microscopic characters of deposit.

7. Skin.

General colour; presence or absence of pigmentation or eruption; nature of "primary lesion" in eruption and of "secondary lesions," if present (pp. 382–386).

Palpate the skin; dryness, smoothness, thickness, elasticity. Character of subcutaneous tissues.

8. Nervous system.

Inquire regarding subjective symptoms (*see* special interrogation).

Investigate state of—

(1) Intellectual functions (intelligence, memory, sleep, coma, delirium, speech, etc.) (Chapter IX.).

(2) Cranial nerve functions (testing them in order) (p. 422).

(3) Motor functions (noting presence or absence of paralysis,

or of abnormal muscular movements, and state of muscular nutrition (pp. 457–468). Electrical reactions of muscles and nerves, if necessary (pp. 486–498).

(4) Sensory functions (including condition of sensibility to touch, weight, temperature, and pain, and the muscle sense) (pp. 468-472); presence or absence of abnormal sensations (p. 472).

(5) Reflexes:—

Superficial Reflexes (p. 473).
Deep Reflexes (p. 478).
Organic Reflexes and Sphincters (p. 483).

(6) Vasomotor and trophic changes. Tache or abnormal flushing. Localised pallor or blueness. Sweating (presence or absence in any locality). Joint changes. Changes in the nails, hair, or skin (abnormal pigmentation, eruptions, atrophies, etc.) (p. 484).

9. The eye.

Appearances seen on ordinary inspection of lids, conjunctiva, cornea, iris, etc. (p. 499).

Use oblique illumination and ophthalmoscopy, noting state of media, refraction, and characters of fundus (pp. 502–516).

N.B.—The fundus of the eye should be reported on in all cases of nervous disease.

The **Ear.**—Examine pinna, meatus, and membrane (using speculum and inflation if necessary) (p. 516).

The **Throat, nose, and larynx.**—Examine larynx (laryngoscopy) and anterior and posterior nares (posterior rhinoscopy) (pp. 521-529), noting any abnormalities.

10. Locomotory system.

Describe any changes in the bones or joints (p. 530).

Diagnosis.

(Prognosis.)

Notes of Treatment and Progress.

(Daily notes in acute cases; in others make a note of progress every three days.)

State on dismissal.

If patient died, add notes of *post-mortem* (if held).

The following special scheme for cases presenting mental symptoms has been drawn up by Dr. Henry Head, and will often be found useful in medical wards:—

(1) GENERAL.

The aspect of the patient as modified by the mental disturbance. General attitude and behaviour.

Any peculiarity in clothing. Does patient tend to strip himself or behave indecently? Can he dress himself? How does he take his food?

General standard of intelligence. Can he read? Can he write? Can he amuse himself with pictures?

Speech as modified by the mental state.

Is he destructive?

Is he dirty in his habits? If so, is it from inattention, or is he actively dirty?

Masturbation, alcoholism, etc.

Is he cataleptic or rigid? Does he make any rhythmical movements or sounds? Restlessness or tremor of the hands? Overaction of muscles of face?

Does he sleep?

Does he tend to wander about the room or house at night?

(2) SENSORY.

Illusions of Sight, Hearing, Smell, Taste. Subjective sensations of touch based upon wrong interpretation of some actual sensation.

Hallucinations

(3) EMOTIONAL.

Exaltation.—Chattering, Shouting, Singing. Excessive sense of well-being. Restlessness or Violence.

Depression.—Crying, Sighing, Moaning. Miserable feeling, either in attacks or continuous. Fear. Is the patient suicidal?

Erotism.—Are the patient's statements coloured by an erotic tone? Give examples.

Religion.—Is the patient's mental state coloured by an extravagantly religious tone?

(4) MEMORY.

Memory of intention, *i.e.* does the patient wish to say or do something and immediately forget the intention? Does patient misplace things?

Memory of recent events.

Memory of remote events [*e.g.* events of childhood].

[If recent memory only is lost, try and find out when the break in memory occurs.]

(5) IDEATION.

Orientation. — Sense of Time and Space. Delusions of identity [*i.e.* does patient mistake those around him for his friends and associates before he entered hospital, or does he imagine they are famous or legendary persons?]. Does he appreciate his surroundings, or does he imagine himself elsewhere than he actually is? Does he invest the acts of those around him with a Secondary or Symbolic meaning? Does he describe actions he has performed, in themselves not impossible or improbable, which, however, did not actually occur? [*e.g.* when in bed with alcoholic paralysis, does he describe the walk he took in the morning, the people he met, etc.?]

Coherence or Incoherence of Ideas.

Delusions of Suspicion.—Continuous or only in attacks.

Delusions of Persecution.—Action of unseen agencies, etc. (especially at night).

Delusions of Grandeur.—Riches, Power, Bodily Strength.

Delusions concerning his *health* or bodily state.

Fears (*unfounded*) in neurasthenia.

CHAPTER II.

General Condition and Appearances.

Before commencing the physical examination, the physician may gather invaluable information from a more general survey of his patient. During the time occupied in asking questions, and even before it, the skilled eye and ear may detect much that has an important bearing on the case. Experience in actual clinical practice can alone educate to this, but some lines may be indicated along which to work.*

One of the first things to observe is the **attitude** of the patient as he lies in bed (decubitus). In health a person lies in any manner in which he feels comfortable—sometimes on his back, sometimes on his side. He changes his position without much difficulty from time to time, and has no hesitation in altering his attitude if he slips from his pillows or feels otherwise uncomfortable. But the stress of disease will often confine his activity in narrow bounds. When fever has run high, or when some other cause has reduced the patient to extreme weakness and dulled his consciousness, he no longer makes an effort to secure a position of comfort, but *passively slips downwards* from his pillows in obedience to the law of gravity, and lies listless, flaccid, and silent even where the resulting attitude is such as to render the act of breathing unnecessarily exhausting.

Almost equally characteristic is the *lateral position* necessitated by some diseases of the viscera, and especially by those of the lungs and pleura. The two main factors in compelling this attitude are, firstly, the greater ease with which respiration can be performed on one side than on the other; and, secondly, the fact that in certain positions the pain is rendered less acute, whilst in others it is aggravated. When these factors co-operate, it is easy to say which side the patient will choose. Thus, in pleurisy with much effusion, where the chief difficulty is the mechanical one of providing sufficient expansion for the uninjured lung, and where pain is slight or absent altogether, the patient will be found lying on the diseased side. If, however, pain be the prominent element, as occurs in the earlier stage of pleurisy, he will best secure easy respiration by lying in the position of least suffering. What this position will be it is not easy to predict, for the pain depends both on the amount of movement and the pressure exerted by the inflamed surfaces on each other. When the inflamed pleura is uppermost its movement is greatest, but its pressure against the chest-wall is least; when it is lowermost the opposite is true; and so when movement is the chief cause of pain the patient will lie on the affected side, but when pressure exerts the greater influence, on the sound one. In either case, however, he confines himself to the selected side, and any change indicates an alteration in the state of the disease.

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* For much fuller information than can be given here, the senior student is referred to Fothergill's "Physiologieal Factor in Diagnosis," and to the writings of Hutchinson, Gairdner, and Laycock.

Another class of patients who prefer one side are those who have a cavity in the lung. When this cavity lies with its aperture below, the secretion flowing from it enters healthy bronchi, and by irritating them maintains a perpetual and most distressing cough. If, however, such a patient turns over, so that the cavity fills before its contents escape, a period of tranquillity is obtained, and though the cough eventually recurs, a larger quantity of secretion is promptly got rid of, and another period of rest secured. When, as frequently happens in phthisis, the secretion is tough and scanty, this symptom is inconspicuous.

Even in health many persons feel more comfortable on one side than on the other, and when ill will often continue to prefer the accustomed attitude; hence the fact that the patient is repeatedly found on one side, although it suggests the propriety of being on the outlook for disease, does not always indicate its presence.

In cases where great demands are made upon the respiratory system, and especially when it fails to respond fully to such demands, the sufferer can rarely lie down in bed, but sits more or less erect and propped up with pillows. To this condition the name of **orthopnœa** has been given. It is common in advanced stages of heart, lung, and kidney disease, and its *rationale* is found in the fact that this attitude permits of freer use of the accessory respiratory muscles, whilst it leaves the diaphragm less impeded by intra-abdominal pressure, and perhaps, also, acts favourably on the intra-cranial venous pressure.* When the abdomen is greatly distended the sufferer cannot flex his thighs without raising the abdominal pressure; at the same time he prefers to sit up rather than to remain in bed, in order that the weight of the fluid may not hinder the descent of the diaphragm. In sitting up, however, he tries to avoid bending his thighs, and therefore he keeps well forward in his armchair, sometimes almost in a kneeling attitude, whilst he rests his head on a table placed before him. In such cases the removal of the ascitic effusion may afford unspeakable relief to the patient.

In abdominal disease, especially when the peritoneum is involved, the aspect is frequently characteristic. The patient lies on his back with a rigidity of attitude and shallow respiration which betoken the pain that any movement produces, whilst one or both legs are drawn up, according as the inflammation is limited to one side or has become more general.

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* *See* Sahli, "Klinische Untersuchungsmethoden," p. 4.

In colic and dysmenorrhœa there is often great restlessness, which contrasts vividly with the fixed attitude of serious inflammation. In renal colic the patient tosses about and tries one position after another in futile search for a posture free from pain, whilst the less acute but more constant dragging pain of renal calculus produces a drooping of the shoulder on the affected side, which is most marked when the patient is erect, but may be present even in bed.

Patients who are attacked by *acute rheumatism* have a peculiar aspect of helplessness, the limbs lying motionless, and the joints being swollen, stiff, and painful.

Various *diseases of the nervous system* produce characteristic attitudes; peculiarly important is that of meningitis, where the neck is bent backwards so that the head seems to bore into the pillow.

When possible, the physician should not only study his patient in bed, but should also see him up and walking. Many very characteristic attitudes, which are of the greatest value in forming a diagnosis, can only be observed when the patient is in the erect posture. Thus the forward stoop, the stiff neck, the tremor, and the fingers flexed at the metacarpal joints and working against the thumb as though engaged in making cigarettes, are as characteristic of paralysis agitans as is the festinant gait.

When the patient is **standing,** observe (1) the pose of the head; (2) the set of the shoulders; (3) the inclination at which the trunk is carried on the pelvis—thrown back in hypertrophic muscular paralysis, in pregnancy, and in massive abdominal tumour, often bent forward when abdominal pain is present; (4) the position of the arms and hands; (5) the outline of the lower limbs.

When the patient walks, any peculiarity in his **gait** must be observed. The more important types of gait are described in Chapter XI., but the student must remember that alterations may not only be due to diseases of the muscular and nervous systems, but that the pain of a gouty toe, or of a blistered heel, or surgical conditions in the ankle, knee, and hip joints, likewise produce characteristic effects.

At least a passing glance should be bestowed on the **dress.** Apart from insanity, where the patient's clothing is frequently dishevelled or grotesque, one may discover indications of a local or general change in his bulk, or his boots may wear unevenly in consequence of some abnormality of gait.

The **general development and nutrition** of the patient demand careful examination. In different types of men very considerable variations must be looked for, and various races differ greatly in breadth of chest. Age also is a factor which cannot be left out of the reckoning, and a proportion between height, girth, and weight that would be natural enough at fifty may be quite abnormal at twenty-one. Recognising, however, that variations must be expected in individual cases, there is still a certain general ratio between height, weight, and chest circumference which has been found to represent the average of a very large number of cases, and may therefore be taken as a rough standard, any wide divergence from which would call for special explanation. The following table is one of several that have been compiled from very extensive statistics.*

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* Hutchinson.

Height.			Normal weight.		Limits of deviation in excess or defect of this which are compatible with good health.
ft.	in.		lbs.		lbs.
5	1	...	120	...	∓ 24
5	3	...	133	...	∓ 27
5	5	...	142	...	∓ 28
5	6	...	145	...	∓ 29
5	7	...	148	...	∓ 30
5	8	...	155	...	∓ 31
5	9	...	162	...	∓ 32
5	10	...	169	...	∓ 34
5	11	...	174	...	∓ 35
6	0	...	179	...	∓ 36

Various attempts have been made, with partial success, to produce a formula which would enable the weight to be estimated when the height and girth are known. One of these, cited by H. Vierordt, is $W = \frac{H\ G}{240}$ kilograms, where W stands for weight, H for height in centimetres, and G for girth in centimetres. If one translates the metric into the more usual British system, and estimates the weight in pounds and the height and girth in inches, the formula becomes $W = \frac{H\ G}{17}$ lbs.

It is important to compare the chest girth taken at the level of the nipples * with that of the abdomen. If in a man below middle age the latter measurement is the larger, it either indicates an undue tendency to fat formation, which may at a later period impair his vitality, or it is due to intra-abdominal disease.

When these measurements have been made, the *nutrition* of the patient is observed. Under this head one notes whether the patient is too stout, is well nourished, or is emaciated. In health there is a fair quantity of subcutaneous fat, the muscles are of moderate size and firm texture, whilst those which have been called into special exercise in the ordinary occupation of the individual under examination may be markedly prominent, and the skin is elastic and neither very moist nor very dry. When nutrition is perverted, the muscles become flabby, and the subcutaneous fat is increased so as eventually to become burdensome to its possessor; or emaciation sets in, owing to the balance between ingestion and excretion becoming deranged, and the waste of tissue exceeding its reparation. Emaciation is thus an important indication of many diseases, especially those which are accompanied with fever.

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* In male subjects.

In estimating the state of nutrition, the observer will take into account the general build of the patient —some are naturally small and slight, others are large and raw-boned; and one also meets with persons who, though possessed of little subcutaneous fat, have well-nourished muscles, whilst others, whose muscles are weak and soft, have an abundant supply of fat in the subcutaneous tissues.

Besides the nutrition of the patient, an attempt should be made to ascertain his **temperament** and, if present, his **diathesis,** since this often exerts a marked influence on the course of his illness. The analysis of temperament is beyond the scope of this work: the senior student will find valuable contributions to the subject in Hutchinson's "Pedigree of Disease."

To the trained observer the **expression** of the patient yields information of the very highest importance, and amongst the factors which determine expression, **the eye** holds the foremost place. Some patients cannot look their doctor in the face, and this tendency to avoid catching his eye is important, as indicating a probability that the information they are about to give lacks truthfulness, and also that they are not to be trusted to obey the instructions which they receive. Sometimes the eyes are restless, following every movement of the attendant, as often occurs in phthisis; at other times they stare vacantly into space, regardless of all that is passing around them, a condition well seen when the consciousness is growing dull. In exophthalmos the eyes are prominent, and show a ring of sclerotic above the cornea; or the prominence may be due to a high degree of myopia. In wasting disease or in profound collapse, such as is found in cholera, the sunken eyes and half-closed eyelids cannot fail to command attention. There are racial differences in the "set" and obliquity of the eyes, and by noting this feature something may at times be learned either of the heredity of a patient or of the tendency to reversion towards a lower type. The arch of the eyebrows may give a hint, where it is either excessive or too slight, of a proclivity to tubercular disease, and an even closer connection seems to exist between the strumous tendency and long, dark eyelashes, coupled with singularly clear sclerotics. More detailed reference is made in a subsequent chapter to important abnormalities in the different structures of the eye, where the student will learn how the conjunctiva and sclerotic tell of tubercle and Bright's disease, of anæmia and rheumatism, of jaundice and of intemperance; and how the cornea foretells an early onset of senile changes in other organs by the appearance of an arcus senilis, or reveals the ravages of syphilis and struma; how the size and mobility of the pupils indicate the existence of disease in the nervous system, or the presence of aneurysm, or it may be only of synechiæ from an old iritis; and how the iris may contain a tubercular nodule, or be muddy and discoloured from iritis.

The **lower eyelids** are puffy and œdematous, especially in the morning, when the patient is suffering from Bright's disease; and a like appearance is often to be noted in patients who are suffering from very severe paroxysms of cough. It is very characteristically present in children affected with whooping cough. The eyelids may also be swollen and inflamed as the result of bug bites. People look "dark under the eyes" when their digestion is out of order, or when fatigued, especially from want of sleep; often women are darker under the eyes during menstruation than at other times.

The **nose** has a sunken bridge in congenital syphilis; the tip is red in some cases of mitral regurgitation, in habitual drunkards, in females with chronic indigestion, and sometimes in purely local conditions. Undue mobility of the alæ nasi may be due to neurosis, or it may indicate obstruction to inspiration, and is in this respect very important to look for in infants. Young persons who suffer from adenoids, and to a lesser extent those afflicted with enlarged tonsils or chronic bronchitis, have pinched noses and open, fishy mouths. The pinching of the nose is due to falling-in of the alæ nasi where they lose the support of the nasal bones whilst the mouth is kept open to reduce the resistance to the entrance of air.

The **lips** are pale in chlorosis and other forms of anæmia; livid and blue in heart disease when compensation fails. A thick, short, and coarse-looking upper lip is often found in association with a phthisical tendency, whilst thin, mobile lips occur in persons of a neurotic temperament, and especially in female invalids whose constitutions are naturally weak, and who have not, nor can ever hope to possess, a large stock of vitality. Herpes on the lip is very often associated with inflammation of the respiratory tract, and is often an early and suggestive accompaniment of pneumonia. When it occurs in a patient who is obviously ill the chest should always be most carefully examined.

The **ears** are often ill-developed in idiots, and sometimes in the insane develop hæmatomata. Of greater frequency is the occurrence of tophi in persons of gouty habit.

The **cheeks** give valuable information regarding the patient's health. In anæmia and aortic disease they are pale; in hectic fever there is a bright circumscribed blush over the malar bones; in the capillary engorgement of plethora they are ruddy and high-coloured, as they also are in many persons who lead an open-air life, exposed to all weathers; in heart disease they are also high-coloured when back pressure has begun to tell on the systemic circulation, but the colour is of a bluish and cyanotic tint, which cannot be mistaken for the rubicund cheeks of plethora. In unilateral chest inflammations, and particularly in pneumonia, the cheek corresponding to the affected lung may be flushed, but if the patient has been lying for some time on one side there is often a difference between the two cheeks, resulting from the pressure of the lower one upon the pillow, quite apart from the presence of disease.

The **form of the cranium** may also indicate points of importance, to which reference is made in Chapter XI.

In addition to the appearance of individual features, the **general expression of the patient** must be noted. Is it animated, apathetic, or has it the absolute vacancy of unconsciousness? Are there wrinkles on the face, or is it smooth; or is one side smooth and the other wrinkled, as one sees it in unilateral paralysis of the seventh nerve? Is the mouth drawn over to one side, and is there any other lack of symmetry between the two halves? The expression may be characteristic of pain, or there may be a placidity resting on the features which gainsays the assertion of a patient that his agony is most severe.

A look of anxiety on a patient's face often presages serious illness at a time prior to the appearance of any other signs or symptoms which would suggest the gravity of the situation. Twitching of the face sometimes results from a nervous habit, at other times it is a symptom of definite disease, of which chorea affords a good example. When **pain** is present, the various features are differently affected, according to its situation. Pain in the head, whether simple headache or of organic origin, causes the sufferer to frown; painful diseases of the chest and abdomen tend rather to affect the expression of the lower part of the face. These signs are of peculiar importance in the case of children who cannot describe their sufferings.* The physiognomy of insanity is often highly characteristic, but descriptions of it must be obtained from special text-books. In serious illness the nose often looks pinched, the eyes look sunken and lustreless, and the chin and malar bones sharp and prominent, owing to the loss of tone which the soft tissues have undergone.

Several types of expression have received special names. Of these the most important are the facies Hippocratica and the typhoid facies.†

In the **facies Hippocratica** the skin is livid or pale, and opaque, the eyes are dull and sunken but remain open, the nose is sharpened, the temples are hollow, the chin is sharp, the mouth is open through dropping of the lower jaw, the ears are cold and shrunken, and the cheeks drawn in. When this facies is associated with abdominal disease there is a red or livid ring around the eyes. The Hippocratic facies is a presage of impending dissolution. The **typhoid facies** is characterised by dull, lustreless eyes, tremor of the lips (with muttering delirium), and a blank, expressionless countenance. Associated with this are found a brown, dry tongue, a rapid pulse, a tendency to sink low in the bed, twitching of the tendons (*subsultus tendinum*), and a constant purposeless picking of the bedclothes.

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* A most valuable contribution to the explanation of these special appearances will be found in Prof. O. Soltmann's "Ueber das Mienen- und Geberdenspiel kranker Kinder," in the "Jahrbuch f. Kinderheilk." Bd. xxvi., p. 206. Students will find an admirable epitome of the main facts in Dr. John Thomson's "Clinical Examination and Treatment of Sick Children," p. 47. Attempts have been made to associate certain lines which appear on the faces of sick children with diseases in special groups of organs. With the exception of a furrow which runs downwards from the ala nasi to curve round the angle of the mouth, and which is not unfrequently present in cases of gastro-intestinal disorder, they are of little or no value for diagnosis.

† *Cf.* Fothergill's "Physiological Factor."

The **state of the skin** where it is exposed must be carefully investigated. In the face we notice especially the **complexion.** This is dependent on two factors—the colour and the transparency of the skin. The most important abnormalities are pallor, yellowness, bronzing, an earthy tint, and a dusky bluish red hue. **Pallor** occurs in various anæmic states, and also when the heart's action is greatly enfeebled, as in fainting or severe nausea. **Yellowness** may be due to pernicious anæmia, when the tint is pale lemon yellow, which contrasts with the whiteness of the sclerotics; or to jaundice, when the skin may be only faintly discoloured, or may be of a dark yellow colour, with excoriations from the scratching that results from the intense itchiness which the bile acids evoke. In jaundice the conjunctivæ and mucous membranes share in the coloration. **Bronzing** is found in Addison's disease, and affects both the skin and the inside of the mouth. An **earthy tint** is common in states of serious ill-health. It sometimes indicates a malarial history; in other instances it is the result of syphilis or of cancer; and in yet others it can be traced to an anæmic condition maintained by continual small losses of blood, such as bleeding piles may involve. This tint is partly due to the altered state of the blood, partly to abnormal opacity of the skin. The **dusky tint** of embarrassed breathing and of advanced heart disease does not demand further notice here.

It is also important to search for **cutaneous eruptions**, some of which—measles and syphilitic rashes, for example—frequently appear first about the roots of the hair, whilst others have equally distinctive situations. Ulcers and scars should also be looked for. The colour and nutrition of the hair, and the dryness or moisture of the skin, must be noted; and if perspiration is present, its amount and situation. The perspiring brow of a rachitic child is very characteristic.

Reference has already been made to the **panniculus adiposus;** but in addition to the presence or absence of fat, morbid conditions may lead to abnormal states of the cellular tissues. The chief of these is the presence of fluid or of air, the former being by far the commoner. When fluid is present, the condition is known as **dropsy,** and there are two varieties of this, which are sometimes described as "hydræmic" and "passive." In hydræmic dropsy, typical examples of which occur in sufferers from Bright's disease, the transudation does not first show itself in the most dependent parts of the body, but in other sites where laxity of the tissues favours its accumulation. Thus in chronic nephritis an early symptom is the œdema of the face, especially below the eyes, which comes and goes, being most noticeable when the patient first rises in the morning. In passive dropsy, however, which is typically present in those cases where pulmonary or cardiac disease produces a backward pressure in the veins, the swelling first appears at the ankles and over the dorsum of the foot, and only gradually mounts to the legs, thighs, and trunk. When the venous obstruction is local, the dropsy is confined to the parts from which the return of blood is impeded. In this way one finds ascites resulting from cirrhosis of the liver, or œdema of an arm when the axillary glands are cancerous and constrict the axillary vein. Œdema of the whole upper part of the body may result from intrathoracic tumours; the writer has seen it follow compression of the superior vena cava by an aneurysm. Dropsy may be recognised by the pallid and glossy appearance of the skin over the swollen part, by its doughy feel, and by the fact that it pits on pressure.

Localised œdema may be due to nervous causes, and is found in certain of the angioneurotic group of diseases.

Subcutaneous emphysema is not common, but when present it can be readily recognised by the crackling sensation which is detected on pinching the part affected.

The **hands** of the patient merit careful observation. Notice the strength of his grip as he shakes hands; this often indicates improvement or the reverse with considerable accuracy. Their general shape should then be noted. Are they stunted as in congenital cretinism, or "spade-like," as in myxœdema; are the joints large, as occurs in rickets and in persons of strumous diathesis? Are they deformed as well, as occurs in rheumatoid arthritis, or swollen and painful, as in acute rheumatism? Sometimes what looks like enlargement of the joints is really due to wasting of the surrounding tissues. When the patient is gouty, the finger joints are often implicated, and nodules, known as **Heberden's knobs**, are formed. These must not be confused with **Haygarth's nodosities**, which are fibrous thickenings found in cases of rheumatism.* Gout sometimes shows itself by producing a contraction of the palmar fascia that prevents extension of the fingers. In nerve disease the skin of the hand may undergo **trophic changes,** becoming thin and glossy; or the vessels may be influenced by vasomotor disorders, and lead to redness or to a pallid and dead-looking state of the fingers. Nerve diseases also produce very characteristic movements or attitudes of the hand, as may be seen in athetosis, tetany, and lead palsy. **Tremor** of the hands is a frequent indication of disease. Amongst the conditions which produce it may be instanced paralysis agitans, multiple sclerosis, certain traumatic neuroses, Graves' disease, uræmia, insomnia, mercurial poisoning, alcoholism, abuse of tobacco, and senile degenerative changes. The methods of studying this symptom are detailed on page 467. In ulnar paralysis the hand becomes deformed by over-extension of the first phalanges, combined with excessive flexion of the rest, so that a claw-like attitude is produced. This is known as the **"main en griffe."** When the muscles of the thenar and hypothenar eminences have undergone atrophy the hand becomes flattened, and thus somewhat resembles that of an ape. In acromegaly and in pulmonary osteoarthropathy there are very characteristic enlargements of the hands, which present a singularly massive appearance. The fingers become clubbed under conditions which produce chronic congestion of the peripheral veins, chief amongst such conditions being respiratory or cardiac embarrassment. Where the congestion is marked the finger tips are blue and cold, and the nails are much curved longitudinally. The nails exhibit longitudinal grooves in gouty persons, in whom also they are often singularly hard and brittle; whilst a transverse furrow is the record of some former interference with the nail's nutrition, and in the absence of a local cause points to some severe constitutional illness. Many persons, especially those of the so-called lymphatic temperament, have moist and clammy hands. These in women frequently indicate excessive leucorrhœal loss, and so aid in directing inquiry towards this subject. Even when the hand is not moist when the patient is seen, the fact that the colour has been sweated out of the palm of the glove may show that the tendency exists.* In infants the movements or position of the hands and fingers will often direct an acute observer to the seat of disease.

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* Considerable difference of opinion exists as to what Heberden really described, but the terms are now generally used in the sense given in the text. Those who desire to consult the originals are referred to Heberden, "Commentaries on the History and Cure of Diseases," London, 1803, 2nd Edit., p .148; and Haygarth, "A Clinical History of Diseases," part ii., Bath, 1805.

The **neck** should always be inspected, and special note taken of any of the conditions about to be described.

1. The state of the **lymphatic glands.**—In syphilis the glands under the upper part of the trapezius are very frequently enlarged. In septic ear diseases the glands below the ear can often be readily felt. Where there are carious teeth, and where there is malignant disease in the mouth, enlarged glands can usually be detected near the angles of the jaw. In scrofulous persons enlarged tubercular glands occur in groups or in long chains beside the sternomastoid, and scars will mark the points where they have suppurated. In lymphadenoma the glands are enlarged and firmly matted together. If enlarged glands are found either in the neck or elsewhere, it is important to observe whether they remain firm and distinct, or become fused together, or whether fluctuation can be detected.

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* Fothergill.

2. The **thyroid gland.**—The existence of any swelling of this gland is important, and its effect on the patient's respiration should be studied. Sometimes such enlargements exercise considerable pressure on the trachea; at other times, particularly if the disease be malignant, the recurrent laryngeal nerves may become implicated. In cases where there is difficulty in determining whether a tumour is connected with the thyroid, much assistance may be obtained from the fact that the gland and any tumour which is connected with it move up and down with the larynx during deglutition.

3. **Unusual prominence of any muscle** or group of muscles in the neck should be described. Such prominence may be bilateral, as of both sternomastoids in emphysema, or unilateral, as in tonic wry neck. A congenital sternomastoid tumour may be present, and, if unrecognised, may lead to much perplexity; whilst various cysts, cold abscesses, or developmental abnormalities may be encountered, Their recognition, however, is rather a question for surgical diagnosis.

4. **Movements of the laryngeal box** are sometimes conspicuous, and may call for explanation.

5. **Rigidity of the neck** may be due to inflammation, to rheumatism, to disease of the spinal column, or to various nervous diseases, whilst spasmodic movements occur in clonic torticollis.

6. Any **bulging of the apices of the lungs** during a fit of coughing, or pulsations seen in the vessels, must be recorded, nor must the existence of aneurysm be overlooked.

7. **Boils and carbuncles** are very frequently situated on the back of the neck. As they are not unfrequently present in cases of diabetes, they should direct the observer's inquiries to the urinary system, and sugar should be tested for.

8. Finally, with respect to the **general shape of the neck**, it should be noted whether it is short and thick, or long and smooth, or "scraggy," or projecting forwards with a prominent larynx. The last form is common in persons with phthinoid chests.

The character of a patient's **respiration** is often of great service in reaching a diagnosis and a prognosis. Under the name of "Extra-auscultation" Professor Wyllie has grouped together the various phenomena connected with respiration which are to be heard, apart from those revealed by the stethoscope, when standing at the bedside of the patient.* In the following scheme his classification is adopted :—

CLASSIFICATION OF THE PHENOMENA OF EXTRA-AUSCULTATION.

I. Obstructive noises in the respiratory passages.

1. In the nose	*a.* Thickened mucosa, or accumulated secretion. *b.* Paralysis of alæ nasi.	
2. In the back of the throat.	*a.* Nasal stertor. The soft palate strikes the back of the pharynx. *b.* Oral stertor. The soft palate strikes the tongue, which has fallen back.	
3. In the larynx	*a.* Swelling of Cords *b.* Paralysis or Spasm of Glottis.	Larnygeal stridor (almost invariably inspiratory.)
4. In the trachea	*a.* Tracheal stridor (leopard growl.) *b.* Tracheal rattle (death-rattle.)	(occur during both inspiration and expiration.)
5. In the bronchi	*a.* Musical sounds (wheezing). *b.* Crepitant sounds.	

II. Cough.
  1. Duration. Single coughs, repeated coughs, paroxysms.
  2. Quality. Resonant or toneless, moist or dry, suppressed or free.

III. Hiccough.

IV. Voice.
  1. Volume.
  2. Quality.

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* *Edinburgh Hospital Reports*, vol. i., p. 48.

Where the *respiratory passages are obstructed* the normal quiet respiratory sound is replaced by more or less noisy breathing. When the obstruction occurs in the nose either from mucus in the meatus or from thickening of the mucosa which covers the turbinated bones, or from paralysis of the alæ nasi, the breathing is sniffing or bubbling in character. When the soft palate is relaxed, and especially when it is paralysed, it prevents the free passage of air between the mouth and thorax, and produces a snoring or stertorous sound. When the rima glottidis is obstructed from any cause, such as spasm or paralysis of the vocal cords or œdema of the larynx, stridulous breathing results. If a polypus or other tumour lie between the cords, there may either be stridor or simply noisy breathing. The trachea may have its airway narrowed by pressure from the outside, as in cases of tumour and especially of aneurysm, when the breathing becomes growling; or mucus may obstruct the lumen, producing a rattling sound. The "death-rattle," which occurs when weakness and insensitiveness combine to prevent any effort at expectoration, is a typical example of the condition. Obstruction in the bronchi gives rise to wheezing and crackling sounds. Sometimes the respiration is sighing; Gairdner is inclined to think that this may indicate fatty degeneration or slight dilatation of the heart. An important division of dyspnœic conditions may be made according as the difficulty in respiration is felt during the inspiratory or the expiratory period. Most cases of obstruction of the air-passages are characterised by **inspiratory dyspnœa,** whilst many of the pulmonary causes of dyspnœa produce **expiratory trouble.** As a common example of the latter one may cite the prolonged expiration of a case of bronchitis with emphysema. The breathing may be characteristic of diseases quite distinct from those of the respiratory system. Examples of this are found in the stertorous breathing of apoplexy, the hissing expiration of uræmia, and the dyspnœa or "air-hunger"* of commencing diabetic coma, which affects both inspiration and expiration.

If **cough** is present, its character must be most carefully noted.† The first thing to observe in this connection is whether the cough consists of independent explosive expirations, or is paroxysmal in character. The former occurs in early phthisis, in granular pharyngitis, and in some forms of nervous irritation; the latter is often found in severe bronchitis, and is very typical in pertussis. One should also notice whether the cough induces pain or nausea, and whether its tone is resonant, or suppressed, or husky. In *common colds* the cough is at first short and dry, but as the quantity of secretion increases, the type becomes more paroxysmal, and the fit of coughing continues till the mucus is expectorated. In *bronchitis* the condition resembles that found in the last affection, but the paroxysms are more severe, and wheezing is often present. When due to *early phthisis*, the cough is frequent, short, and sharp, and is described as *dry* because there is no rattling of mucus associated with it. Later in the disease, when the caseous masses are breaking down, secretion is much more copious, and the cough becomes moist and paroxysmal. In severe cases actual vomiting may be induced. A *nervous cough* generally has the character of single, short, dry explosions, repeated at intervals, and a similar type is produced by irritation of the peripheral nerves, whether the source of the irritation be found in a disordered stomach or threadworms in the rectum, or be due to disease in the ear or to the discomforts of teething, or take origin in the nerves of the pregnant uterus. Local conditions in the *throat* may be the cause of most troublesome and persistent coughing, and a careful observer will not fail to look for granular pharyngitis when the patient complains of constant hawking, or for a relaxed and trailing uvula, more particularly when the cough starts the instant the patient lies down.

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* Küssmaul.

† The student will observe that two elements must be discriminated in a cough. The first of these is the *explosive element*, due to the sudden opening of the valve formed by the false cords and surrounding structures; the second is the *vocal element*, due to the rush of released air between the true cords (*vide* Wyllie, *loc. cit.*).

In *pleurisy*, *pneumonia* (associated as it often is with more or less pleurisy), and in *pleurodynia*, the cough consists of solitary dry, hacking, expulsive efforts, suppressed as much as possible to prevent unnecessary pain, but repeated frequently. In *laryngitis* and *croup* the cough may be simply noisy, but more often is either husky or stridulous. When the lumen of the trachea is encroached upon by a *mediastinal tumour* or an *aneurysm* there is generally a very resonant, brassy cough, aptly compared to the cry of a gander. When once heard, this is almost sufficient to clinch the diagnosis without further examination.

In *hysteria* the cough is often loud and barking, and gives the impression of being produced with the view of attracting attention. Such a cough is sometimes associated with hysterical aphonia. *Pertussis*, when it is fully developed, is distinguished by a most characteristic cough. There is first a long-drawn, almost stridulous inspiration, then a series of short, sharp, expiratory coughs, which follow each other with extreme rapidity. The face turns dark and the veins grow prominent, the child clings firmly to any support it can find, so as to give full play to the accessory muscles of respiration, and when at last the fit of coughing ends it is followed by a long-drawn whooping inspiration. The severity of the paroxysm induces vomiting, and sometimes causes evacuation of the bladder and bowel.

Hiccough, which results from spasmodic contraction of the diaphragm, is a common enough disorder. It may be due to trivial causes, such as an attack of indigestion; but it also occurs, and that most persistently, in many serious illnesses, when the symptom may become one of considerable gravity. Thus, if it be met with in a patient whose kidneys are affected, and especially if the occurrence in such a case follow the passage of instruments to relieve stricture, there is ground for serious apprehension as to the issue.

The **voice,** as well as the cough, should be studied. The chief points to observe are its strength, whether it is clear or husky, or whether aphonia exists. The voice may be nasal either through habit or in consequence of obstruction in the upper airways. A distinction should be made between open and stopped nasal tones, the former resembling the sound produced when the mouth is kept shut during phonation, the latter that heard when one speaks whilst holding the nose.

Temperature.—The hand laid upon the skin gives a certain amount of information as to the temperature, especially if there be no perspiration; but a far more accurate guide is found in the *thermometer*, whose use should never be omitted.* In taking the temperature the following practical points must be attended to:—

1. The thermometer must be accurate and of good quality. To insure accuracy, it should be compared with a standard instrument. In Britain this is

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* Just because the hand takes account of moisture as well as of the actual temperature, it may convey information which the thermometer fails to impart—*e.g.* the "pungent" dry heat of early pneumonia is most characteristic.

done at Kew, and certificates are issued which state the error of each individual instrument. In process of time, however, and particularly if the thermometer has been recently made, molecular changes occur in the glass which tend to make the reading too high. Such changes are slight, and seldom attain a value of any clinical importance, though if great accuracy is necessary a fresh comparison should be made every two or three years. Also, if the bulb of the thermometer be made too thin, the glass will yield to pressure, and the patient may either purposely or accidentally compress it so much as to make the mercury reach to four or six degrees above the actual temperature.*

2. The thermometer must be kept in position long enough to allow the mercury to reach the body temperature. Generally it is well to exceed the period which the instrument professes to require.

3. In adults the temperature is taken in the mouth or in the axilla; in young children the thermometer should be placed in the fold of the groin, and the thigh flexed on the abdomen, or it may be inserted into the rectum. The temperature of the mouth and rectum is generally at least half a degree higher than that of the groin or axilla, but in old people the mouth temperature is often too low, and less trustworthy than that of the axilla. When taken in the latter situation, care must be exercised to keep the part as free as possible from perspiration, both during the observation and for a few minutes before it. Moreover, the arm should be drawn to the side

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* Mr. Hicks, Hatton Garden, London, E.C., supplies an aseptic thermometer which can be cleansed in plain water, as there are no divisions or figures on the stem to harbour dirt or germs. The graduations are engraved on the case, into which the thermometer is inserted after it has been used, and this possesses the further advantage that they are not obliterated by the repeated cleansings of the instrument, as so frequently happens in those of the usual pattern.

for a short time before the thermometer is inserted, that the skin may not have been chilled by exposure to the air.

4. Before inserting the thermometer, make an invariable rule of washing it in lotion or in cold water, and see that the mercury is well shaken down; wash it again before replacing it in its case. In Great Britain the Fahrenheit scale is used, on the Continent the Centigrade.*

The temperature *should be taken at fixed times*, twice daily when possible, and at shorter intervals when fever fluctuates or runs high. Times that are convenient, and that fairly represent the daily conditions, are at 9 a.m. and 7 p.m. In health the temperature has a daily range of from one to two degrees Fahrenheit, being lowest in the small hours of the morning, and gradually rising to attain its principal maximum somewhere about five or six p.m. Age exercises a rather marked influence on the temperature. In children it varies greatly with their time of life, and trivial causes produce great fluctuations. On the average, it is about half a degree higher than in adults. In the very old it is also slightly higher than in middle life, unless the circulation is weak, when the temperature may be considerably lower.

In diseased conditions marked **deviations from the normal temperature** are often present. Temperatures may be classified as follows: –

Normal† ...	98°—99°F. or 36·5—37·2°C.
Sub-normal ...	below 98°F. or below 36·5°C.
Collapse ...	below 96°F. or below 35·5°C.
Febrile ...	above 99°F. or above 37·2°C.
Hyperpyrexia	above 107°F. or above 42·0°C.

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* For a comparison of the two scales, *see* Appendix 4, p. 593.

† The figures given are merely approximate. Wunderlich allows a rather wider range for normal temperatures than those stated above, and taking the mean normal temperature at 37°C. would allow a range of from 36·25° to 37·5°C.

By consecutive observations, taken at suitable intervals, it is easy to determine whether an abnormal temperature is constantly present, or only occurs at intervals. When the temperature rises quickly, the patient feels chilly in consequence of the incomplete response of the vasomotor mechanism to the new conditions, and in marked cases **rigors** occur. If, however, the temperature remains continuously high, the rigor gives place to a feeling of heat, coupled with thirst, headache, and a rapid pulse. This is known as **pyrexia**, or fever. If after fever the temperature falls rapidly, or if during the fever the extremities are chilled, the patient suffers from **collapse**, when the pulse is small, the features are pinched, the skin is moist with a clammy sweat, and the patient suffers from a sinking sensation and from nausea.

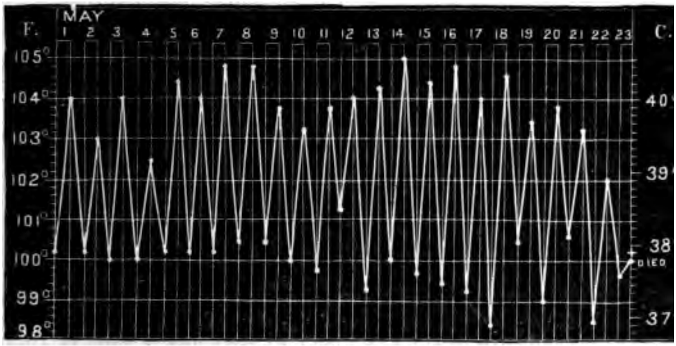

Fig. 1.—Remittent fever (hectic). Case of phthisis. (*After Finlayson.*)

There are three principal **types of fever**—the continued, the remittent, and the intermittent. When fever does not fluctuate more than about a degree and a half (Fahrenheit) during the twenty-four hours, and at no time touches the normal, it is described as **continued.** When the daily fluctuations exceed two degrees, it is known as **remittent** (Fig. 1); and when fever is only present for several hours during the day it is called **intermittent.** In remittent fever the evening temperature is usually higher than the morning one, but in some cases, not unfrequently in phthisis, this type is *inverted*, and the "remission" occurs in the evening, whilst there is a morning "exacerbation." When a paroxysm of intermittent fever occurs daily, the

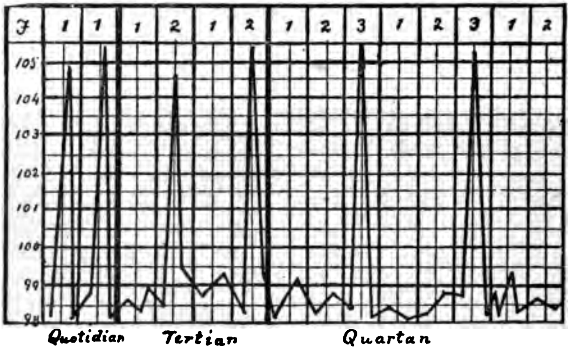

Fig. 2.—Intermittent fevers.

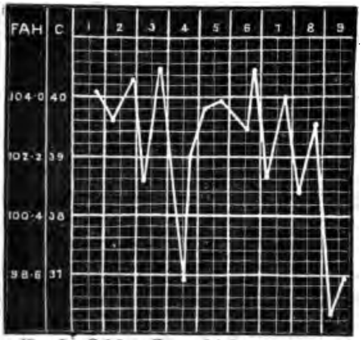

Fig. 3.—Crisis. Case of lobar pneumonia. (*After Wunderlich.*)

type is said to be "**quotidian**"; when on alternate days, "**tertian**"; when two days intervene between consecutive attacks, "**quartan**" (Fig. 2). A "**double tertian**" is the name applied to a daily fever when the paroxysms occurring on the first, third, fifth, and following odd days differ from those of the second, fourth, sixth, and following even days in hour of appearance, in severity, or in character.

The **course of a fever** is divided into three stages—the initial or pyrogenetic, "*stadium incrementi*"; the stage of full development, or "*fastigium*"; and the stage of termination, or "*stadium decrementi.*" When the fever ends rapidly it is said to resolve by "*crisis*" (Fig. 3); when gradually, by "*lysis*" (Fig. 4). Not seldom crisis is preceded by a short but marked rise of temperature, accompanied in many cases by delirium; it is sometimes followed by collapse.

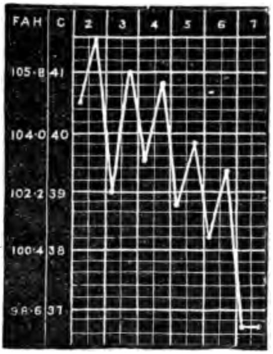

Fig. 4.—Lysis. Case of broncho-pneumonia. (*After Wunderlich.*)

In the study of any case of fever the points which require to be observed are whether the type is one of apathy and indifference, or of restlessness and twitching; whether, and if so how far, the sensorium has been involved; what the height of the temperature is, and what its course has been; what are the rate and character of the pulse; whether the skin is moist or dry, or exhibits any eruption; and which of the viscera or secretions are characteristically affected. The explanation of these points may be found in works on medicine, but their true significance can only be learned at the bedside.

CHAPTER III.

ALIMENTARY SYSTEM AND ABDOMEN.

SECTION I.—THE MOUTH, THROAT AND ŒSOPHAGUS.

The mouth.—For the examination of the mouth the patient should be placed facing a good light. If artificial light is used it should be thrown into the mouth by means of a reflector. A piece of candle wrapped in blotting-paper and held in front of a bright spoon forms a good extemporised light and reflector.

Lips.—Note the colour of the lips. They are blue in cyanosis, pale in anæmia. Note the presence of any crusts, fissures, or ulcers. The lips should be everted in order to permit of an examination of their inner surfaces. Herpes of the lips is often seen in inflammatory conditions of the air-passages and lungs, especially in croupous pneumonia.

The teeth.—The *temporary teeth* are cut in the following order:—

First.—The two lower central incisors, sixth to eighth month.

Second.—The four upper incisors, eighth to tenth month.

Third.—The lower lateral incisors and all the front molars, twelfth to fourteenth month.

Fourth.—The canines (upper first), eighteenth to twentieth month.

Fifth.—Posterior molars, at two to two and a half years.

The *permanent teeth* appear as follows:—

First molar at six years.

Central incisors at seven years.

Lateral incisors at eight years.
Bicuspid (anterior) at nine years.
Bicuspid (posterior) at ten years.
Canines at eleven to twelve years.
Second molars at twelve to thirteen years.
Third molars at seventeen to twenty-five years.

The following table shows the relations of the permanent and temporary teeth :—

		M.	Bi.	C.	I.	I.	C.	Bi.	M.	
Temporary	Upper	2		1	2	2	1		2	20
	Lower	2		1	2	2	1		2	
Permanent	Upper	3	2	1	2	2	1	2	3	32
	Lower	3	2	1	2	2	1	2	3	

The presence of any irregularity or defect or carious disease in the teeth should be noted. It should be observed whether there is any exposure of their roots, or whether they are surrounded with tartar. Grinding of the teeth leads to bevelling of their edges; this is specially found in young children. The presence of "Hutchinson's teeth" is important as affording evidence of congenital syphilis. In this condition the two central upper *permanent* incisors are at a higher level than the adjoining teeth; they are rounded in section and slope inwards below, they are broader nearer the gum than at the crown, so as to be peg-shaped, and they present a semi-lunar notch at their ends. They are usually discoloured as well. In the same condition the molars tend to be dome-shaped.

The gums.—Their colour should first be noted. In lead poisoning a blue line can often be observed running along the gum near the insertion of the teeth, and especially, perhaps, on the gum between the teeth. In copper poisoning a greenish line can sometimes be seen in a similar position. The gums may be swollen and spongy in scurvy. They are sometimes retracted or show ulcerations or hæmorrhages. Ulcers and hæmorrhages may also be observed in the buccal mucous membrane in various conditions.

The tongue.—Ask the patient to protrude it. Note if it is put out in a straight line. Observe its size and shape, whether broad or pointed. Look for tremulousness of the whole tongue and for fibrillary twitching of it. Note in the dorsum (1) its colour: is it pale, red, or discoloured? (2) Is it dry or moist? (3) The presence or absence of fur; the colour and distribution of the latter should be noted. (4) The character of the papillæ. (5) The under surface of the tongue—a small ulcer on the frænum is sometimes seen in persistent coughing, and particularly in whooping cough. Lastly (6), observe the edges of the tongue. Look for ulcers, indentations of the teeth, etc., on them.

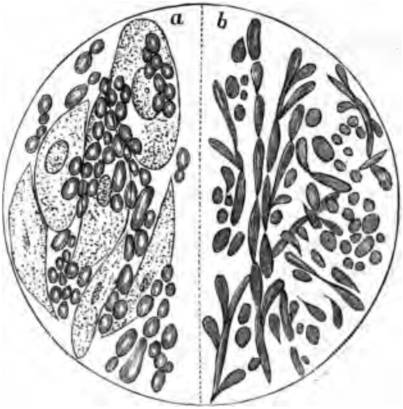

Fig. 5.—*a*, Scraping from a patch of thrush; × 800. *b*, Culture of saccharomyces albicans (monilia candida)—the fungus of thrush; × 800.

The presence of **thrush** may sometimes be observed on the surface of the buccal mucous membrane, especially in children. It presents the appearance of small white points or patches raised somewhat above the surrounding surface, which is sometimes redder than normal. Patches of thrush are very apt to be mistaken for small milk curds. They may be distinguished by the fact that milk curds can be easily detached, while thrush patches can only be removed with difficulty, and when removed are apt to leave behind a raw surface. To search for the fungus (saccharomyces albicans) a small piece of the patch should be scraped off and examined in a drop of glycerine. A quantity of epithelial *débris*, along with bacteria and leucocytes, will be seen, and mixed up with these the filaments of the fungus. These consist of long but unequal segments, each usually possessing a refractile nucleus at each end (Fig. 5).

The palate, fauces, and pharynx.—Introduce a tongue depressor, and note first the general colour of the soft palate, fauces, and pharynx; observe any abnormal degree of pallor or redness. The yellow tinge of jaundice often lingers long on the soft palate, and in commencing measles a patchy redness can be made out very early in the same situation. Note the presence of any ulcers or mucous patches on the palate, fauces, or tonsils. Look carefully at the **tonsils**, noting any enlargement of them. Yellowish or greyish points or patches may sometimes be seen on their surface. Try whether these can be wiped off, leaving a sound surface, as is the case with accumulated follicular secretion, or whether removal leaves behind a raw surface, as happens with the false membrane of diphtheria. Note always whether or not the soft palate and uvula show any similar spots or patches. Next look at the **pharynx.** The presence upon its surface of a number of flat adenoid swellings, somewhat resembling sago grains, is so common as to be almost a normal appearance. In granular pharyngitis these are much increased. A few dilated venules can also be frequently observed. Note the presence of any pus or excess of mucus on the surface, and the existence of any ulceration. In retro-pharyngeal abscess the posterior wall of the pharynx is bulged inwards. Sometimes this can be more easily made out by palpation.

The breath.—The character of the breath may be noted at this stage. If it is offensive, ask the patient to breathe out first through the nose only, and then through the mouth, and observe whether the odour is present on both occasions or not. This affords an indication as to whether the source of the odour is in the nose or mouth only, or whether it is lower down than either. If the odour proceeds from the nose, make a rhinoscopic examination (p. 526), looking especially for the presence of a foreign body or for evidence of atrophic rhinitis or other local disease. Bad teeth, ulcerations of the gums or mucous membrane, and enlarged tonsils, accompanied by retention and decomposition of secretion in their follicles, are the commonest sources of offensiveness in the mouth.

In gangrene of the lung the breath has a putrid smell. In bronchiectasis, also, it has a peculiarly offensive odour only to be recognised by experience. Fœtor due to pulmonary conditions is best brought out by asking the patient to cough.

Slighter degrees of offensiveness may be due to gastric disorder or to prolonged constipation.

In uræmia the breath has a urinous or ammoniacal odour. In diabetes it is sweetish, like new-mown hay. In cases where diabetic coma is impending, the odour becomes ethereal. Various drugs—*e.g.*

turpentine, creosote, paraldehyde, etc., impart their characteristic odours to the breath, while in the case of patients who are taking bismuth a garlicky odour can often be observed. Iodides produce a peculiar fœtor.

THE ŒSOPHAGUS.

[illegible] teeth to
cardia [illegible] c.m.

Special anatomy.—The œsophagus is from 9 in. to 10 in. long. It begins opposite the cricoid cartilage, and ends opposite the ninth dorsal spine. It is crossed by the left bronchus between the fourth and fifth dorsal vertebræ.

Exploration of the œsophagus.—This is done in cases in which there are signs of stricture. It is best carried out by means of a stomach tube. The latter should never be passed, however, unless one has first excluded the possibility of the existence of an aneurysm. It should also be avoided in cases where there has been any recent hæmatemesis.

It is best to use a long, red rubber stomach tube, No. 20 or 21 in the English scale. It should be at least a yard long—not too thin in the wall, rounded at the end, and with at least one large eye. Previous to being used, it should be thoroughly cleaned, and then dipped in hot water; oil is unnecessary.

If the pharynx is very irritable, it may be anæsthetised with cocaine, either by painting or by giving the patient a pledget of wool to suck, soaked in a 5% solution, and directing him to keep the saliva at the back of the mouth. After the lapse of five minutes, one can proceed to pass the tube.

The patient should be sitting up, with the head slightly bent forwards. His mouth is open, but the tongue *not* protruded. The physician grasps the tube in his right hand, and passes it back in the middle line to the posterior wall of the pharynx. It is not usually necessary to introduce a finger into the mouth

Deglutition murmur
7 sec.

in order to guide the tube, as there is no real danger of entering the larynx. The patient is then told to swallow, and the tube is "payed out" until it reaches the stomach, or until it is permanently arrested. Whilst this is going on the patient should be told to breathe freely through his nose.

During this manipulation one has to look out for the following: (1) *Pain* on passing the tube; its site should be noted. (2) The presence of an *obstruction*. If the tube is arrested, one must not conclude all at once that a stricture exists. Frequently the tube is seized by a muscular spasm of the œsophagus. On waiting for a moment, however, this always passes off. If a permanent obstruction be discovered, one has to attempt to localise it. The commonest sites for a stricture are—(*a*) At the entrance; this is 6 in. from the incisor teeth. (*b*) Where the œsophagus is crossed by the left bronchus; this is 8 in. to 9 in. from the teeth. (*c*) At the cardiac orifice; this is about 17 in. from the teeth.

It is interesting to note that these are also the positions in which some normal narrowing of the œsophagus exists.

(3) One has to look for *diverticula*. The existence of such should be suspected when the tube passes very regularly at one time, but it is obstructed at another.

The presence of any blood on the tube after its withdrawal is an indication of the presence of ulceration. Sometimes, also, fragments of new growth can be detected in the eye of the tube.

Auscultation of the œsophagus.—This is done in order to note the presence or absence of the sound produced by swallowing. In order to auscultate the œsophagus in the neck, the stethoscope should be placed at the left side of the trachea. In the upper part of the thoracic course of the œsophagus (as far as the 6th dorsal vertebra) it should be placed just to the *left* of the dorsal spines, and below this just to their *right.* The patient is told to take a mouthful of water, and to retain it until told to swallow. When he swallows, one hears a noise similar to that heard in one's own ear on swallowing saliva. The higher up one listens, the louder is this sound. If an obstruction is present, the sound is either not heard at all below that point, or it is greatly delayed.

SECTION II.—THE ABDOMEN.

Anatomy.—The natural lines on the surface of the abdomen are (1) the linea alba; (2) the lineæ semilunares; (3) the lineæ transversæ.

The *linea alba* is often selected as the site of puncture in tapping the abdomen. The structures lying behind it, from above downwards, are (*a*) the left lobe of the liver, extending to about three fingers' breadth below the ensiform; (*b*) part of the stomach, unless when empty; (*c*) the transverse colon, reaching as low as the umbilicus; (*d*) coils of intestine covered by omentum; (*e*) the bladder when distended, and the uterus when pregnant.

The *linea semilunaris* runs from the lowest part of the seventh rib to the spine of the pubes. It is about three inches from the umbilicus, but lies farther out when the abdomen is distended. The gall bladder lies just to the outer side of the linea semilunaris of the right side.

Of the *lineæ transversæ* one is opposite the umbilicus, another at the ensiform, and a third midway between these points. In addition to these markings, the abdomen has been artificially divided into **regions** by means of vertical and horizontal lines.* The vertical lines are drawn upwards from the mid-point of Poupart's ligament on each side. The transverse lines are (1) the infracostal, drawn across horizontally at the level of the lowest points of the 10th costal cartilages, and (2) the bi-iliac, between the most prominent points of each iliac crest. Nine regions are thus marked off in three vertical rows. Those in the middle row are, from above downwards, the epigastric, umbilical, and hypogastric, and in each lateral row we have the (right or left) hypochondriac, lumbar, and iliac regions. The contents of these regions are exhibited in the annexed table.

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* In describing the regions of the abdomen we have followed Quain. We should like, however, to point out that for clinical purposes the *interspinous line*, drawn between the two anterior superior iliac spines, has many advantages over the bi-iliac.

The umbilicus is 1¼ in. to 1½ in. above the level of the bi-iliac line, and lies opposite the upper part of the 4th lumbar vertebra. Its position is far too variable for it to be a trustworthy landmark.

The aorta bifurcates about ¾ in. below and slightly to the left of the umbilicus, the iliac arteries running in a line drawn from that point to the middle of Poupart's ligament.

The cœliac axis arises at a point 4½ in. to 5 in. above the umbilicus, and the renal arteries about an inch lower than the cœliac axis.

GENERAL EXAMINATION OF ABDOMEN.

The patient should be lying on his back in a good light. The abdomen is exposed by turning down all the bedclothes except the inner sheet. The nightshirt should then be drawn up, and, lastly, the sheet folded down a little above the level of the pubes. These details are of especial importance in the case of female patients. Before beginning the examination of the abdomen, make sure that the bladder is empty. If necessary, a catheter must be passed.

Inspection of abdomen.—Look first at the general contour of the abdomen. Is it of normal

Right Hypochondriac. Most of R. lobe of liver. Hepatic flexure of colon. Part of R. Kidney.	Epigastric. Part of R. lobe of liver. Whole of L. lobe of liver (usually). Gall bladder. Part of stomach, including orifices. 1st and 2nd parts of duodenum. Pancreas and upper end of spleen. Parts of the kidneys. Suprarenals.	Left Hypochondriac. Part of L. lobe of liver (sometimes). Part of stomach. Splenic flexure of colon. Tail of pancreas and most of spleen. Part of L. kidney.
Right Lumbar. Part of R. Kidney. Ascending colon. Part of ileum (sometimes).	Umbilical. Part of R. and sometimes both kidneys. Most of transverse colon. 3rd part of duodenum. Coils of jejunum and ileum. Part of mesentery and gt. omentum. Part of stomach.	Left Lumbar. Part of L. kidney (sometimes). Descending colon. Part of jejunum.
Right Iliac. End of ileum. Cæcum and vermiform.	Hypogastric. Coils of ileum. Upper part of rectum and sigmoid loop Bladder in children and (if distended) in adults. Gravid uterus.	Left Iliac. Coils of jejunum and ileum. Sigmoid flexure.

fulness, is it swollen or protuberant, or is it sunken or retracted? If there is any bulging, note if it be general or local. General fulness, it has been epigrammatically remarked by Professor Wyllie, may be due to "fat, fluid, or flatus." If one were to venture to improve upon this, it would be to add "fœtus" as a possibility in the case of women. It must further be remembered that a *new growth* may also be a cause of general abdominal tumidity. The mode of distinguishing these conditions will be considered when we come to ascites. In general bulging it should be noted whether the distension is most marked in the antero-posterior or in the transverse diameter.

If the bulging be merely local, observe in which zone it is situated. Is it above or below the level of the umbilicus, and in which of the abdominal regions is it most marked? Lastly, note if there is any movement to be seen in the swelling, either along with or independently of respiration.

Pulsation in the epigastric region is a phenomenon which may be noticed on abdominal inspection, apart from any bulging in that region. The causes of it are (1) distension of the right ventricle (see p. 117); (2) venous pulsation of the liver; (3) aortic pulsation. The last is a condition which is very frequently observed in nervous subjects, especially women. The cause of it is obscure. It may be distinguished from right ventricle pulsation by being situated somewhat to the left of the middle line, by the fact that it can usually be traced downwards towards the bifurcation of the aorta, and by its being not quite synchronous with the apex beat, but somewhat delayed. (4) Transmitted pulsation from a tumour overlying the aorta. (5) Aneurysmal. This is, perhaps, the least common cause of epigastric pulsation. The pulsation in this case is expansile, a fact which can best be elicited by placing the narrow ends of two stethoscopes one on each side of the swelling, and observing if the other ends move apart at each impulse.

The **movements of the abdominal walls** should be studied. Normally, they bulge during inspiration, and fall in again during expiration. In paralysis of the diaphragm the reverse holds true; sometimes the paralysis is unilateral, in which case one side of the abdomen will move naturally. Cessation of movement of the abdominal walls is a valuable sign of peritonitis.

Sometimes **peristaltic waves** are visible through the abdominal wall. This is especially apt to be the case in chronic intestinal obstruction. The coils of intestine above the constricted part then stand out prominently. From this a definite "pattern" of abdominal tumidity results, depending on the site of the obstruction. If, for example, there be a constriction at the ileo-cæcal valve, the distended coils of small intestine may often be observed standing out in the centre of the abdomen one above the other, so as to form a "ladder pattern." On the other hand, if the obstruction be low down, say in the sigmoid flexure, the pattern of tumidity is one in which the periphery of the abdomen is chiefly affected. A dilated stomach may also stand out as a prominent tumour in which peristaltic waves are visible. The direction of such waves should always be noted. If absent, they can often be elicited by flicking the surface with a wet towel, or by merely sharply tapping it with the finger. Peristaltic waves in the stomach run from left to right; those in a distended transverse colon from right to left. This may sometimes be of diagnostic value.

Attention should next be paid to the **surface of the abdomen.** In great distension the surface is smooth and glossy. Striæ (white lines in the epidermis) should be looked for; they indicate former distension. Note any distension of the surface veins, and endeavour to ascertain in what direction the blood in them is flowing. In obstruction of the inferior vena cava, the inferior epigastric veins are full from the establishment of a collateral circulation. In such cases also a large *lateral vein* can be seen running up about the mid-axillary line, and thus establishing a communication with the tributaries of the superior vena cava. In portal obstruction a number of distended veins may often be seen radiating out from the umbilicus. To this appearance the term "caput medusæ" has been applied. It is due to establishment of a connection between the portal and parietal veins by means of the round ligament. *Pigmentation* of the abdominal wall is sometimes important. Along the middle line it forms the linea nigra—one of the signs of pregnancy. Note the appearance of the *umbilicus*. Is it depressed, level with the surface, or bulging? Is there any excoriation around it? Lastly, one should never omit to look at the usual sites for any evidence of hernia.

Palpation of the abdomen.—The patient should be on his back, with the knees drawn up, and the shoulders a little raised. He should be told to keep the mouth open and to breathe quietly, or his attention may be diverted by conversation. The hand of the physician must be warm. Ordinary palpation should be performed with one hand only. In order to gain the confidence of the patient's abdominal muscles, the hand should be allowed to rest for a moment on the surface of the abdomen before palpation is actually commenced. Each region should be palpated systematically. Poking with the finger tips should be avoided, the best movement being a gentle one from the metacarpo-phalangeal joints. During expiration the receding abdominal wall should be followed by the fingers, and a gentle rotatory motion of the finger tips may then be carried out. It often enables one to feel the deeper structures better than one can do by simple pressure. In examining the lateral regions of the abdomen, bimanual palpation is often of service. The physician should sit or kneel by the bedside. One hand is placed posteriorly in the interspace between the last rib and the crest of the ilium. The other is placed over the abdominal wall in front. The posterior wall is then pushed up against the hand in front, so that any structure lying between the two hands can be distinctly felt. The secret of the method consists in keeping the front hand as still as possible. This procedure is of special value in the examination of the kidneys.

The first thing to notice in palpation of the abdomen is the degree of tension of the walls and of *resistance* experienced. Normally, the abdomen has an elastic or doughy feeling only to be learnt by practice. In disease the resistance may be increased. It should be observed whether this increase is general or local. General peritonitis produces a great increase in the resistance from a reflex contraction of the muscles of the abdominal wall. Local increase in resistance is very frequently due to localised peritonitis, and is often of great diagnostic value. Palpation of the normal abdomen is painless. If tenderness is elicited, its exact extent and point of maximum intensity should be noted. Anything of the nature of a tumour should be carefully felt for. In doing this, confusion is apt to be brought about by the recti. The thickening produced by parts of these may easily simulate a tumour. If this source of fallacy be suspected, try if the fingers can be got under the edge of the muscle, and feel if it thickens as the patient raises himself in bed.

If it be decided that a tumour is really present, one has first to determine whether it is situated inside the abdomen or in the abdominal wall. Try, therefore, to move the abdominal wall from side to side over the tumour. If the growth be intra-abdominal, this can usually be done without difficulty, unless it has contracted adhesions to the parietal peritoneum. Try also to grasp the tumour and to make the fingers meet, as it were, under it. This can usually be accomplished in the case of tumours situated wholly in the abdominal wall.

Supposing the tumour to be intra-abdominal, the first question to be settled is—Where is it growing from? and, especially, is it coming up out of the pelvis, or is it truly abdominal? To decide this the edge of the hand should be pushed back about an inch below the umbilicus, and in the direction of the prominence of the sacrum. One can then feel whether the tumour is passing down into the pelvis or not. The size and shape of the tumour should next be noted, and the nature of its surface—whether smooth or nodular. The presence or absence of fluctuation should then be investigated.

The *mobility* of a tumour is a very important point to determine. The directions in which it can be moved should be noted, and whether it is influenced by respiration. The latter is a point of special value. Tumours connected with the liver and spleen move freely with respiration. Tumours of the kidney may be slightly movable; those connected with the other abdominal organs do not move with respiration at all unless they have contracted adhesions.

In palpating the abdomen, the existence of *splashing* or *gurgling* at any points should be looked for. Splashing is often found over a dilated stomach, but is only of diagnostic value if it can be elicited some hours after the swallowing of food. Gurgling is produced by the passage of gas and fluid through constricted parts of the alimentary tract. It may thus be felt at the pylorus, especially if stenosed, or over strictures of the intestines.

Finally, the *umbilicus* should be examined. In malignant disease of the liver the umbilicus often becomes early infiltrated, and this sign has proved of great diagnostic value. The infiltration can often be recognised by its producing a "mooring" of the umbilicus—just as a scirrhus of the mamma does of the nipple.

In conclusion, we have to remark that in obscure cases of abdominal disease palpation in the knee-elbow position, and under an anæsthetic, should never be omitted.*

Percussion of abdomen.

Percussion should be carried out in the same manner as will be described for the chest. We would point out, however, that in abdominal percussion the "flicking" method is extremely serviceable in detecting slight degrees of dulness—*e.g.* in making out the lower edge of the liver. In carrying out this method the forefinger of the left hand is placed firmly on the abdomen, with the palmar aspect uppermost, and is then sharply "flicked" with the middle finger of the right hand. Percussion of the normal abdomen yields a resonant note throughout, except in the regions of liver and splenic dulness, or over a full bladder. The percussion pitch of the hollow viscera depends on two chief factors—

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* Examination of the patient in a hot bath has recently been recommended as of even greater value than the use of an anæsthetic in obscure abdominal cases, besides being safer. The patient gets into the bath at 100° F., and the temperature is rapidly raised by the addition of very hot water until 110° F. is reached. Complete relaxation of the abdominal wall is usually brought about at this temperature in five or ten minutes, but in some instances one may require to go up to 120° F. before the desired result is obtained.

(*a*) The depth of the air space.

(*b*) The tension of the containing wall.

As these two factors are of almost equal importance, and as each of them varies greatly in the same viscus at different times, the reader will readily understand that it is a mistake to dogmatise about the relative pitch of the note yielded by the various hollow viscera. The presence of free gas in the peritoneal cavity causes the normal liver and spleen dulness to disappear.

If any abnormal dulness be detected, the chief point to be determined regarding it is whether it is constant in position or shifts with alterations in the position of the patient. This will be more fully discussed when we come to speak of ascites.

Hydatid cysts yield on percussion a special kind of vibration called the "*hydatid thrill.*" To elicit it three fingers should be placed over the cyst, and the middle one firmly percussed, the percussing finger being allowed to rest for a moment after each stroke. An "after-thrill" will then be experienced in the two adjacent fingers. It should be added that the sign is absent in about half the cases of hydatid cyst.

Auscultation of abdomen.

Auscultation is not of much service in the examination of the abdomen. It is best to carry it out by means of a binaural stethoscope. In the region of the stomach one may listen for swallowing sounds, bubbling and splashing (to be described later), or for abnormal conduction of heart sounds. Elsewhere one may hear friction sounds—from the presence of lymph on the surface of the peritoneum. One may listen over aneurysms to detect a bruit, and over suspected enlargement of the uterus for the presence of the uterine souffle or fœtal heart sounds. The latter are best

Earliest recognization of jaundice in a Blood · by separating [illegible]

heard (in normal presentations) at a point midway between the umbilicus and the left anterior superior spine.

The examination of the abdomen by the combined auscultation-percussion method will be referred to later.

In cases of general abdominal swelling **measurement** should never be omitted, as it affords a valuable index of the progress of the case. The circumference may be taken at the level of the umbilicus or at the point of maximum distension.

The examination of cases which are believed to have fluid in the peritoneal cavity or **ascites** calls for special consideration. In cases in which the fluid is sufficient to cause general distension, the conditions for which one is apt to mistake it are, as we have seen, fat in the abdomen and abdominal wall, gas in the intestines or free in the peritoneum, and new growths. Fluid gives, of course, a dull note on percussion. The dulness is not always absolute, however, owing to the transmitted resonance of subjacent bowel. When the fluid is free and not sufficient to fill the whole abdomen, its upper limit is more or less horizontal, but may show irregularities owing to the fluid running up into "bays" between coils of bowel.

Free fluid is also distinguished by the fact that it shifts its position with that of the patient. If he be turned over on his side and time given for the intestines to float up, it will be found that the uppermost flank is now resonant, while the height of the dulness on the lower side has risen. If the fluid be very small in amount, it is a good plan to turn the patient on his hands and knees. A dull area then appears around the umbilicus from accumulation of fluid there.

The "*transmitted thrill*" is another important physical sign of fluid in the peritoneum. It is elicited by placing one hand over the lumbar region of one

[illegible]

[illegible] 141) [illegible] and [illegible]

side, the patient being on his back, while the opposite lumbar region is sharply tapped with the fingers of the other hand. A distinct impact will be felt to pass from one hand to the other. As a not dissimilar impulse is apt to be transmitted through the abdominal wall, especially if fat, it is always well to get an assistant to place the edge of his hand firmly in the middle line of the abdomen while percussion is being made. This damps down any vibrations transmitted by the wall. Where the amount of fluid is large, the vibrations are visible as well as palpable. On the whole we consider that the results of simple percussion afford the most trustworthy evidence of the presence of ascites.

Fat is to be distinguished by taking the abdominal wall between the hands and pinching it up. *Gas* is distinguished by the results of percussion. Of *new growths*, ovarian tumour is, perhaps, most liable to be mistaken for ascites. An ovarian tumour, however, causes an antero-posterior bulging of the abdomen, while in ascites the bulging is mainly lateral. In ovarian tumours, also, the dulness is central, and does not change with the position of the patient; in ascites the chief dulness is in the flanks, and it shifts, as we have seen, when the patient is moved. Lastly, in ascites, the umbilicus is flat or bulges out, while in ovarian tumours it is drawn upwards.

SECTION III.—THE ABDOMINAL VISCERA.

One may now pass to the examination of the viscera contained in the abdomen, beginning with the stomach.

THE STOMACH.

Special anatomy (Plates I., II.).

The stomach is situated in the left hypochondriac and the epigastric regions. Its cardiac orifice lies

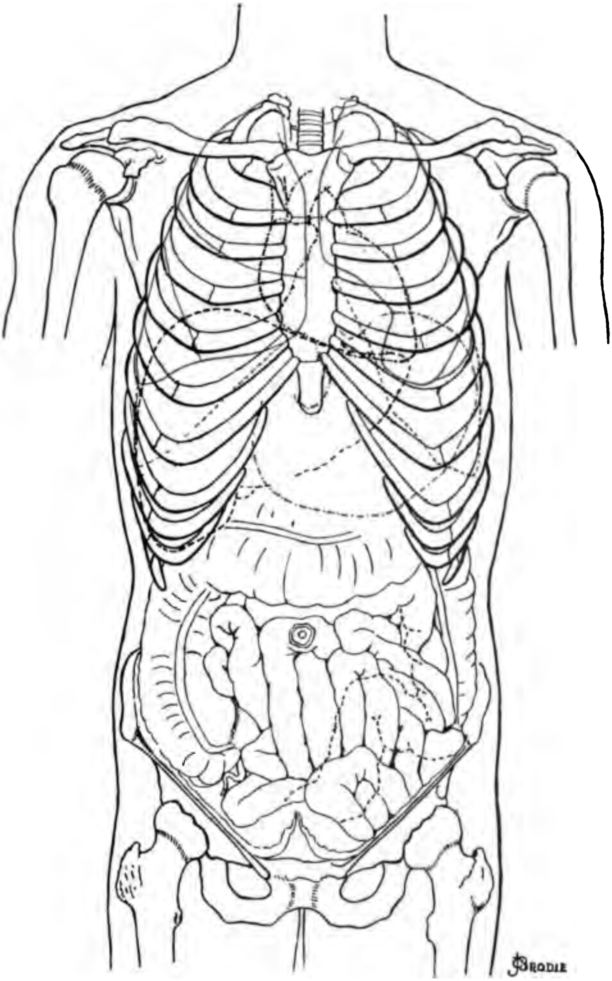

PLATE I.—VISCERA OF THORAX AND ABDOMEN, AS SEEN FROM THE FRONT. Scale: 1 = 5·6. (*After Luschka.*)

[To face p.

Methods for Stomach 2.4.

(1) Instruments

2) [illegible]

3) [illegible]

[illegible])

[illegible]

[illegible]

behind the seventh left costal cartilage, one inch from the sternum and four inches from the surface. The pyloric orifice is surprisingly close to it, being about one full hand's breadth below the base of the xiphisternum and one finger's breadth to the right of the middle line. It passes considerably farther to the right, however, when the organ is distended. It is usually under cover of the liver. About two-thirds of the stomach is under cover of the ribs, the fundus reaching, in ordinary circumstances, as far up as the fifth rib in the mammary line. It is, therefore, somewhat behind and above the apex of the heart. Only a small part of the body of the stomach and of the pyloric region is in contact with the anterior abdominal wall. The exact position of the great curvature varies. *Under normal conditions it should never be lower than the level of the umbilicus; usually it is considerably higher.*

The physical examination of the stomach is chiefly concerned with the determination of its size. It is by no means easy to be sure of the exact dimensions of the stomach, owing in part to its position, in part to the fact that the amount of gas contained in it varies greatly from time to time. The fact that it is in direct contact with the transverse colon, which yields a very similar note, also adds to the confusion.

Recourse is frequently had to *inflation* of the stomach with gas in order to overcome some of these difficulties.

The best method of inflating the stomach is to pass a stomach-tube provided with a glass mouthpiece. One distends the stomach either by blowing down the tube or by connecting it with a Higginson's syringe or bicycle pump. One can clamp the tube when a sufficient degree of distension has been attained.

Inflation should never be practised if there has been any recent bleeding from the stomach, or if one has reason to suspect the existence of a gastric ulcer. It should also be avoided in feeble subjects and in those in whom the heart is in any way embarrassed.

Inspection of the stomach region is included in the general examination of the abdomen. If dilated the organ may possibly be seen standing out even before inflation. It may form a tumour in any part of the abdomen except the upper portion of the epigastric region. If it be dislocated downwards, the outline of the lesser curvature may be visible. Peristaltic movements of the stomach wall have already been referred to (p. 57).

Palpation of the stomach.—Note if there be any tenderness felt on palpating the stomach, and define its point of greatest intensity. Examine for tumours. The commonest of these is a pyloric new growth. Tumours of this region are characterised by their great mobility. They may be felt in, or pushed into, any region of the abdomen. Lastly, try for *splashing.* To make out this, sit at the left side of the patient with one hand over the left lower ribs behind; with the other placed over the front of the stomach make short, sudden dipping movements. If "splashing" be elicited it will be partly heard and partly felt.

Distinct splashing elicited three hours after a meal, especially if it can be made out below the level of the umbilicus, is very suggestive of a dilated stomach.

Percussion of the stomach.—Three boundaries of the stomach can be made out by percussion: (1) Between stomach and lung; (2) between stomach and liver; (3) between stomach and colon.

The last is that which it is most important to determine, and should be examined first. It is by no means always easy to define the lower border of the stomach exactly. This is because the transverse colon may give an almost identical note with it. Use light percussion. The "flicking" method succeeds very well here. Begin low down near the pubes and percuss upwards just to the left of the middle line. The lower border of the stomach should be reached at least a finger's breadth above the umbilicus.

The limit between the lower edge of the lung and the upper part of the stomach is made out in a similar way. It is better to percuss from stomach to lung. The usual line of demarcation between the two runs in a slightly arched manner from the sixth costal cartilage in the parasternal line to the ninth rib in the mid-axillary line. The area of stomach resonance which is bounded above by this line and by the anterior edge of the spleen, and below by the left costal margin, is called **Traube's space.** It covers that portion of the stomach which is in direct contact with the chest-wall. We have seen that the fundus of the stomach extends above this under cover of the lung as high as the fifth rib in the nipple line. It cannot, however, be satisfactorily percussed out.

The demarcation between the stomach and liver is made out by percussing lightly from the stomach towards the liver margin. It is not of much importance in diseases of the stomach.

We would repeat that for the diagnosis of dilated stomach the position of the great curvature is of most importance. If this be found to be below the umbilicus, while the lesser curvature is in its normal position, then the existence of dilatation is certain. It is confirmed if the stomach note extends much across the middle line towards the right.

The position of the boundaries between stomach and lung and stomach and liver depends as much upon the condition of the lung and liver as upon the stomach itself. Thus in fibroid contraction of the lung more of the stomach is exposed than is normal, and its area is therefore apparently increased. Cirrhosis of the liver may produce a similar apparent extension of stomach area. On the other hand, effusions into the pleura or enlargements of the liver coverup the stomach and cause a diminution in its area of resonance. Hence in the former condition Traube's space is much diminished (*see* p. 260). The possibility of a dislocation of the whole organ downwards must never be lost sight of. In such a case the outline of the lesser curvature may often be seen standing prominently out, especially when the organ is inflated. If the distance between the greater and lesser curvature is more than 10 cm., dilatation exists.

The **combined percussion-auscultation method** often gives valuable aid in mapping out the stomach. To carry it out proceed as follows:—Place the end of a stethoscope over the stomach, either somewhere in Traube's area or, better, in the angle between the xiphoid cartilage and the left costal margin. Then percuss lightly by flicking the skin near the stethoscope. A characteristic note is heard. Then, still keeping the stethoscope in the same situation, start percussing near the pubes, and percuss up towards the umbilicus until a note similar to that first heard is made out again. That indicates that the edge of the stomach has been passed. One can now, by percussing in different directions, make out the limits of the organ all round. The method depends on the fact that the aërial vibrations set up under the percussing finger resound all through the organ, and therefore reach the stethoscope placed over any part of it. Previous inflation of the organ is not required. Be careful not to use too strong a percussion stroke, or vibration may be set up in adjoining viscera.

Auscultation of the stomach area may detect peritoneal friction over it, or the crackling

Pallor [illegible]

[illegible] [illegible] linden

(4) Character of wall

Kinks [illegible]

1. [illegible]
2. [illegible]
3. Pressure [illegible]
4. [illegible]
5. [illegible]
6. [illegible]

Enlargement [illegible]

1. [illegible]

trans [illegible]

also

2. [illegible]
3. [illegible]
4. [illegible]
5. [illegible]

[illegible]

1. [illegible]
2. [illegible]
3. [illegible]
4. [illegible]

[illegible]

1. [illegible]
2. [illegible]
3. [illegible]

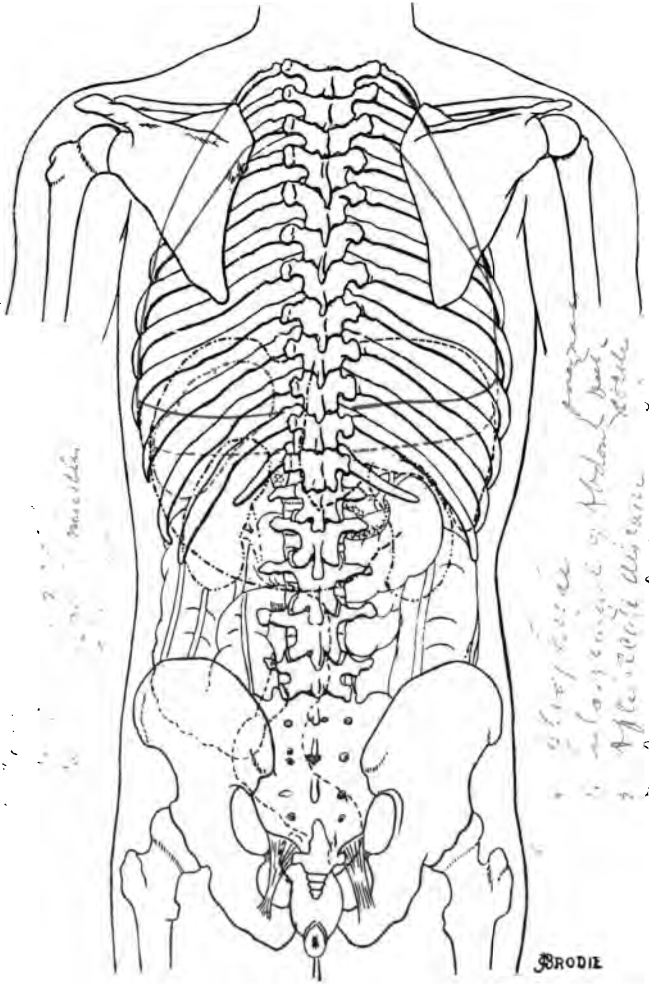

PLATE II.—VISCERA OF THORAX AND ABDOMEN, AS SEEN FROM BEHIND. Scale: 1 = 5·6. (*After Luschka.*)

[To face p. 69

1. [illegible]
2. enlargement of abdom. [illegible]
3. [illegible] disease
3. Swelling of [illegible]

due to the bursting of fermentation bubbles in the interior. Heart sounds and murmurs are sometimes heard loudly over the stomach as over a resonating chamber. The deglutition sounds are of no diagnostic value.

The chemical investigation of the stomach is considered in Section IV. (p. 83).

THE LIVER.

Special anatomy (Plates I., II., III.).—The liver lies chiefly in the right hypochondrium. Its left lobe extends across the epigastric region, but does not pass more than two inches to the left of the sternum. Above, the liver reaches almost to the nipple; below it extends to the costal margin. The lower border passes obliquely upwards from the ninth right to the eighth left costal cartilages, crossing the middle line somewhat above the mid point between the base of the xiphoid and the umbilicus.

The gall bladder is situated just internally to the ninth right costal cartilage, and immediately to the outer side of the right rectus muscle.

Inspection of the liver is of little value. Any visible swelling, fulness, or pulsation should be noted. The edge of the liver can sometimes be seen when the organ is enlarged. It forms a sharp line which moves up and down with respiration.

Palpation of liver.—The lower edge should first be felt for. In order to do this, place the hand flatly on the abdomen, with its edge towards the costal margin and just to the outer side of the rectus muscle, the reason for going so far out being to avoid the upper septum of the rectus sheath, which is apt to be mistaken for the lower edge of the liver. Then depress the edge of the hand slightly so as to push up a fold of skin, and ask the patient to take a long breath. If the edge of the liver is palpable, it will be felt to ride over the edge of the hand. Trial, of course, must be made at different levels before it is decided that the edge cannot be felt. The edge of the liver cannot, or can only very rarely, be felt in health. It moves down from two-fifths to three-fifths of an inch with inspiration. The character of the edge should also be noted—whether it is smooth or irregular, thickened or sharp. If in doubt whether what is felt be really the liver edge, feel for the fissure of the gall bladder, and, towards the middle line, for that produced by the round ligament.

The *surface of the liver* in the epigastrium should then be felt in the usual way. Any tenderness should be noted, and whether it is localised or general. The character of the surface should be made out. Is it smooth, as in waxy disease, or nodular, as in carcinoma? In the latter condition the centres of the nodules will often be found to be umbilicated. Care must be taken not to confound little irregularities which are frequently present in the upper parts of the recti with irregularities on the surface of the liver. Liver friction (due to perihepatitis) can sometimes be felt. It is usually best made out over the posterior surface of the organ between the vertebræ and the mid-axillary line.

Heaving pulsation of the whole organ can best be appreciated by placing one hand over the lower right ribs behind and the other over the organ in front.

The *Procédé du Pouce*, or "thumb method," is a special means of palpating the lower border of the liver devised by Glénard.* The patient lies on his back, the legs extended and the shoulders slightly raised. The physician sits on the edge of the bed facing him. The hands are placed as shown in Fig. 6. The fingers of the left hand push forward the right loin, the middle finger being placed just below

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* "Les Ptoses Viscérales," p. 913.

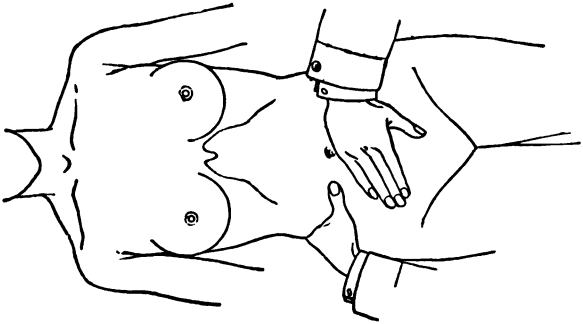

Fig. 6.

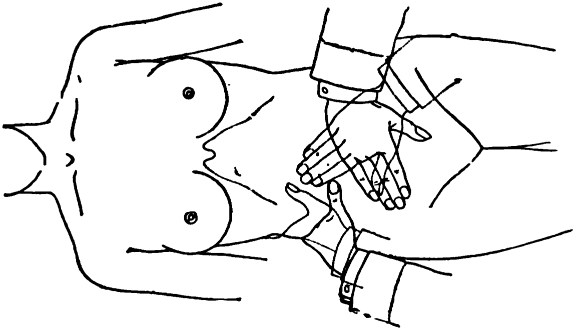

Fig. 7.
To illustrate the *Procédé du Pouce* (p. 70).

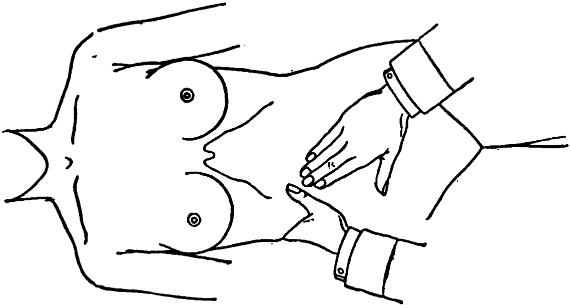

Fig. 8.

the last rib. The right hand depresses the anterior abdominal wall in the hypogastric and right iliac regions, and pushes the coils of intestine up under the liver. In performing this part of the manipulation the fingers of the right hand move in the arc of a circle, as shown in Fig. 7. The left thumb now depresses the abdominal wall just below the supposed situation of the edge of the liver, whilst at the same time the patient makes a deep inspiration. As the edge descends the pulp of the thumb should slip upwards, outwards, and forwards, so as to come in contact with it.

This method is specially serviceable for the detection of ptosis of the liver or a floating (Riedel's) lobe.

Percussion of the liver.—The following table shows the normal percussion limits of the liver (Fig. 23):—

		Middle line.	Mammary line.	Mid-axillary line.	Scapular line.
Upper limit.	Deep dulness.	Blend with heart dulness.	4th space.	7th space.	9th space.
	Superficial dulness.		6th rib.	8th rib.	10th rib.
Lower limit.		Hand's-breadth below base of xiphoid.	Costal margin or somewhat above or below it.	10th space.	Blends with kidney dulness.

Procedure.—The patient should be lying down in percussion of the anterior and lateral aspects; sitting up or standing for the posterior aspect.

To make out the deep dulness, use heavy percussion—two fingers, if necessary. Begin high up—say about the second rib—so as to get a good lung note, and percuss down from rib to rib till impairment is detected. Then repeat the process, going from space to space instead of from rib to rib. Percuss in this way down the mammary, mid-axillary, and scapular lines.

The upper limit of liver dulness in the middle line cannot be distinguished from the heart dulness. To map it out, draw a straight line from the apex beat to the angle where the right edge of the heart and the deep liver dulness meet. To make out the upper limit of superficial dulness, percuss lightly down the same lines. The upper limit of liver dulness forms an almost horizontal line around the chest.

In defining the lower edge of the liver, use very light percussion, and pass upwards. The "flicking" method does well. Another good plan is to percuss with three fingers of the right hand held in a row. Very slight degrees of dulness can often be more easily detected by this device.

The exact position of the lower edge of the liver is extremely variable. Usually it coincides with the costal margin in the mammary line. It may be considerably above or below this, however, without there being any pathological change in the organ. Its position in the middle line is also very variable. As a rule, it is situated about a hand's breadth below the base of the xiphoid.

In percussing the surface of the liver where it is not covered by lung, it should be observed that the organ has a certain degree of *resistance* or *resilience*. The normal amount of this can only be learnt by practice. If the organ be enlarged or congested, its resistance to percussion is increased owing to its being more firmly pressed against the chest wall.

The liver may be displaced, enlarged, or diminished.

Displacement may be either upwards or downwards. Upward displacement may occur from

Mc Carey Detroit Med.

Bar Frank [illegible]

tumours, etc., in the abdomen pushing the liver up. Downward displacement may be brought about by dilatation of the right ventricle of the heart, right pleural effusion or emphysema of the lungs, or, more rarely, by new growths below the diaphragm. When the liver is dislocated downwards, the rounded upper surface of the left, and part of the right, lobe can usually be made out crossing the epigastrium. A displaced liver also does not move freely with respiration, while a liver which is merely enlarged does.

One must distinguish between real enlargements and diminutions of the liver and those which are apparent only.

This enlargement of the liver may be simulated by consolidations of the base of the right lung, or by effusion into the right pleura. Downward enlargement may be simulated by accumulation of fæces in the transverse colon.

Real enlargement may be due to waxy disease, congestion, fatty infiltration, hypertrophic cirrhosis, new growths, leucocythæmia, abscess, or hydatids.

A hydatid cyst in the liver often produces an enlargement of the organ upwards rather than downwards.

Diminution of the liver may be simulated by the organ being covered up by an emphysematous lung, or by the colon passing up between it and the abdominal wall. The latter is a rare condition. It should be suspected if the lower limit of liver dulness varies very much at different points. Real diminution occurs in cirrhosis and in acute yellow atrophy.

The **gall bladder** is examined by palpation and percussion. It cannot be felt unless distended. It may then form a smooth, pear-shaped tumour, situated just to the outer edge of the right rectus muscle. It can be moved freely from side to side

Halitosis / Bad breath

Intra Intestinal

1. Caries [illegible]
2. Follicular tonsillitis – Bronchiectasis [illegible]
3. [illegible]
4. Gastritis [illegible]
5. Constipation [illegible]
6. [illegible]
7. Anaemia [illegible]
8. Syphilis [illegible]
9. [illegible]
10. Cancer of [illegible]

Spice [illegible]

1. [illegible]

[illegible]

Liver Enlargement
1. Congestion active / Passive
2. Neoplasm
3. Abscess
4. Fevers
5. Cirrhosis – Hyp of Art / – Hypertroph
6. Rickets
7. Poisoning
8. Leucaemia
9. Cysts
10. Hodgkins D
11. Fatty
12. [illegible]

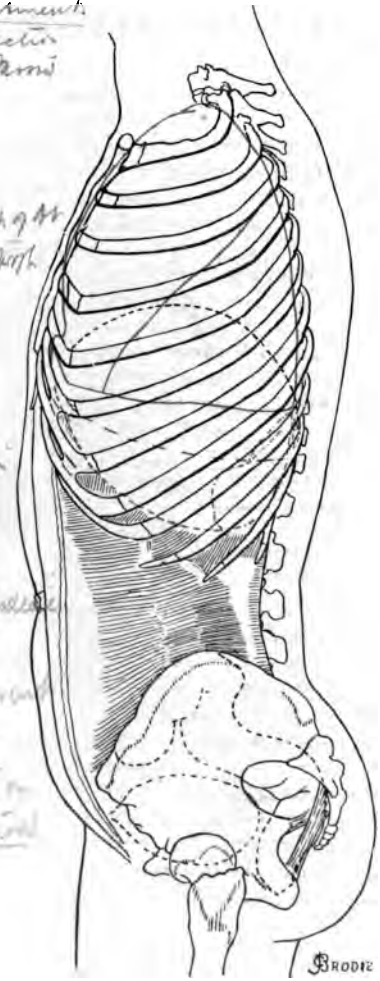

PLATE III.—VISCERA FROM THE SIDE. The lower limit of the lun shown both after full inspiration and full expiration. The line of refle of the pleural sac is also shown. Scale: 1 = 5·6. (*After Luschka.*)

[To face p. 7

round a point opposite to the ninth costal cartilage. It also moves with respiration. Sometimes gall stones can be felt in it. If there be many of these they produce on palpation a sensation resembling that produced on feeling a bag of nuts.

On percussion, a distended gall bladder forms a dull area, projecting out from the liver dulness towards the umbilicus, but usually continuous with it. Sometimes, however, the transverse colon comes to lie across the neck of the gall bladder, so as to separate it from the liver. When this occurs, diagnosis of the tumour is apt to give trouble. To this point we shall recur when we come to the examination of the right kidney (p. 79).

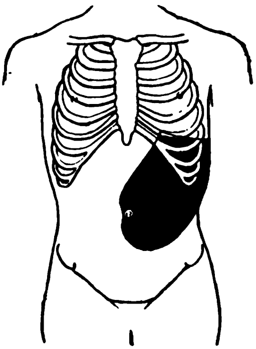

Fig. 9.—Enlargement of Spleen.

THE SPLEEN.

Special anatomy (Plates II. and III.).—The spleen lies in the left hypochondrium. It is bounded above by lung, elsewhere by stomach and intestine. Its lower end rests upon the costo-colic fold of peritoneum. It lies along the ninth, tenth, and eleventh ribs, being partially separated from them by the diaphragm and lower edge of the left lung. Its upper end is opposite the ninth dorsal spine, and reaches to about 1½ in. from the middle line. Its lower end comes as far forward as the mid-axillary line.

Inspection of the spleen.—If much enlarged, the spleen may form a visible tumour in the left side of the abdomen, which moves with respiration (Fig. 9).

Palpation of the spleen.—This is really the most important method of investigating the spleen. If one can exclude dislocation, then a spleen which is palpable may safely be pronounced to be enlarged, and it is never safe to diagnose enlargement of the spleen unless it *is* palpable.

There are two methods of feeling for the spleen—

(1) Stand or sit at the left side of the patient as he lies on his back. Keep the hand flat on the abdomen, and depress the fingers a little, so as to push up a fold of skin near the left costal margin opposite the tenth cartilage, and get the patient to take a long breath. The edge of the enlarged spleen will be felt to come up against the finger tips, and to ride over them, as it were.

(2) Go to the right side of the patient. Place the fingers of one hand behind in the space between the ends of the tenth and eleventh ribs. Place the other hand over the left hypochondrium, with the fingers slightly tucked in under the edge of the costal arch. With the posterior hand tilt the spleen forwards while the patient inspires. The edge of the organ will then be felt against the fingers of the other hand.

The edge of the spleen is sharp and usually quite smooth. Notches can often be felt in it, but by no means invariably. It is important to note (1) that the anterior border of an enlarged spleen is always directed downwards and inwards, and (2) that there is always a slight space between the posterior edge of the spleen and the erector spinæ, into which the fingers can be dipped. Occasionally the spleen enlarges upwards only. This may happen where the costo-colic fold is abnormally well developed, and keeps the organ up. For the detection of such a condition one must have recourse to percussion.

Sometimes, on the other hand, when the spleen gets very large it pushes down the costo-colic fold and then drops down itself. A spleen which at one time crossed the middle line may cease to do so by dropping down in this way, though the organ is really larger than ever.

Percussion of the spleen.—The anterior part of the spleen can be defined whilst the patient is lying on his back. For the posterior part one of two positions is advisable—he may either sit up, with his left hand supported on the top of his head, or he may be semi-prone, resting chiefly on the right scapula, with the left arm behind the head. He should not be altogether on his right side, else the spleen falls away too much from the surface of the body. The organ is most sharply defined when percussed with the chest either in full inspiration or full expiration. It is least clearly made out when the chest is in an intermediate position.*

The limits of the normal spleen cannot always be defined by percussion. It may be borne away so much from the surface, owing to extreme arching of the diaphragm, or it may be so covered up by lung, that it is impossible to be sure of its exact limits.

Procedure.—Define the *anterior* edge by percussing lightly along the tenth rib, beginning near the costal edge. The splenic dulness (absolute) should be reached at the mid-axillary line.

The *lower edge* is defined by percussing lightly upwards along the posterior axillary line or slightly behind it. The lower edge of the spleen should be reached about the lower border of the eleventh rib.

To make out the *upper* and *posterior* borders, heavier percussion is required. Percuss vertically downwards about midway between the posterior axillary and scapulär lines, beginning at about the level of the angle of the scapula. The lung note will become impaired at the upper edge of the ninth rib, indicating that the upper limit of the spleen has been reached.

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* *See* Buttersack (*Zeit. f. klin. Med.*, 40, p. 244).

The *posterior border* is defined by percussing along the tenth rib, beginning near the middle line. The splenic dulness is reached at about 1½ in. from the vertebral spines. This border is not always easy to make out.

By joining together the different points defined above, an oval area will be mapped out, which measures about 3 in. in its long diameter and 2 in. transversely.

Extension of splenic dulness may be simulated by effusions into the left pleura, or consolidation of the base of the left lung; by the presence of fluid in the stomach, or of fæcal accumulation in the colon.

Enlargement of the spleen occurs in acute fever (especially typhoid), in waxy disease, malaria, various blood affections, etc.

Auscultation over the spleen may be practised to detect the existence of friction. The latter occurs in perisplenitis and over the surface of splenic infarcts.

THE KIDNEYS.

Special anatomy (Plate II.).—Each kidney lies partly in the epigastric, partly in the hypochondriac region. The right kidney lies partly in the lumbar region as well. As regards their relation to the anterior abdominal wall, the kidneys are higher up than one is apt to suppose. The lower end of the right kidney is fully 1 in. above the umbilicus, the left is about ½ in. higher. The lower end of each is about 3 in. from the middle line.

As regards their posterior relations, about one-third of each kidney lies above the last rib. The upper end of the right kidney is at the level of the eleventh dorsal spine, whilst its lower end reaches to about 1 in. above the iliac crest. The left kidney is about ½ in. higher.

Palpation of the kidney.

Procedure.—The patient must be on his back with the knees slightly flexed and the shoulders raised on a firm pillow. The lumbar region must be quite flat on the couch, not arched forward. Sit or kneel beside the patient on the side to be examined. Place one hand upon and below the last rib behind, the other immediately below the costal margin in front. It is best to have the right hand in front when examining the right kidney, and the left hand in front when examining the left. The posterior hand should press the loin forwards, while the other hand pushes the anterior abdominal wall backwards, upwards, and outwards. The kidney will then be felt between the two hands if it is at all enlarged or displaced. Even in health (provided the patient is not too fat) the lower part of the organ can often be felt.

Should this manipulation not succeed, the patient must be asked to take a long sighing inspiration. If the front hand follows up the receding abdominal wall as the air leaves the chest the kidney will be caught between the two hands.

The kidney moves very slightly with respiration. An exaggeration of this normal mobility, so that the organ slips up and down like a pea in a pod, constitutes "*movable kidney.*" This must be distinguished from "*floating kidney,*" in which the organ moves freely forward so as to "float" towards the anterior abdominal wall, as well as moving vertically and laterally.

A floating right kidney is very apt to be mistaken for a distended gall bladder, and *vice versâ.* The shape, size, and consistence of the tumour may be apparently identical in the two cases. One point of distinction is that while a distended gall bladder can be temporarily pushed back from the abdominal wall, yet it always tends to spring forward again. It is therefore always in evidence. It is not so with a floating kidney; the latter often disappears for a time, and can only with difficulty be got hold of again. Another point of distinction is that a kidney can be pushed down towards the pelvis and held there even during forcible expiration, whilst the gall bladder moves upwards again during the expiratory act. The different relation of the colon to the kidney and to the gall bladder should also be remembered.

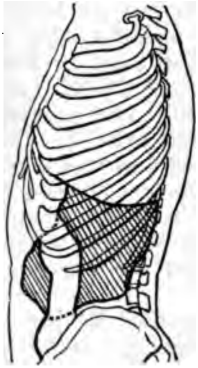

Fig. 10.—Showing colon crossing a tumour of the kidney. (*After Sahli.*)

An enlarged left kidney may be mistaken for the spleen. The points of distinction are: (1) That the spleen has a sharp edge. The edge of the kidney is *always* rounded. The existence of a sharp edge in any abdominal tumour excludes the kidney at once. (2) There is no space between the posterior border of the kidney and the erector spinæ, as there is in the case of the spleen; and (3) the colon lies between the kidney and the anterior abdominal wall, but not over the spleen (Fig. 10).*

It is impossible to determine the size of the kidney by means of percussion.

An enlarged kidney tends to bulge forwards. Perinephric abscesses, etc., bulge backwards.

* If one is in doubt whether the colon lies in front of the tumour or not, it is often a help to inflate the large bowel by means of a rectal tube connected with a Higginson's syringe or bicycle pump.

THE INTESTINES.

Special anatomy.—The small intestine occupies chiefly the umbilical and hypogastric regions; the large intestine, the peripheral zone of the abdomen. The ileum joins the colon at a point 2 in. internal to, and somewhat above, the right anterior superior iliac spine. The apex of the cæcum corresponds to a point a little to the inner side of the middle of Poupart's ligament. The base of the vermiform appendix usually lies opposite a point 1½ to 2 in. from the anterior superior spine along a line drawn from that spine to the umbilicus. This is sometimes called McBurney's point, because he has shown that in the majority of cases of appendicitis that is the point of maximum tenderness as determined by the pressure of one finger.

The splenic flexure of the colon lies behind the stomach, the hepatic lies under cover of the liver. The former is at a somewhat higher level than the latter. The transverse colon passes across the abdomen in a slightly curved direction, the lower part of the curve reaching to about the umbilicus.

Examination of the intestines by **inspection** and **palpation** has been described earlier in the present chapter under the general examination of the abdomen (p. 54).

Percussion of the intestines.—The notes yielded by the small and large intestine cannot be satisfactorily discriminated. The combined percussion-auscultation method can be used to map out the colon in the same way as was described for the stomach. One should place the stethoscope near the splenic or hepatic flexure, and percuss close to it. Then one may begin at what is presumably beyond the periphery of the gut, and continue to percuss towards the stethoscope until one recognises the characteristic note.

Rectal examination.—Place the patient in a good light and in a semi-prone position—*i.e.* resting on the left breast with the right thigh and knee well drawn up, the inner aspect of the right knee resting on the couch. Draw aside the glutei and inspect the region of the anus, noting the presence of any eruption, of external hæmorrhoids, etc. Smear the right forefinger with vaseline and fill the nail with soap. Pass the finger slowly and gently through the anus, directing it slightly forwards at first. Note the degree of resistance offered by the sphincter; this shows whether the latter is normal, spasmodic, or relaxed.

Once the anal canal is passed, direct the finger slightly backwards and upwards, asking the patient to bear down a little at the same time. The finger can then be swept round and the whole inner surface of the rectum explored.

Two folds of mucous membrane will be encountered (Houston's folds), one opposite the prostate in front, the other higher up, near the middle of the sacrum, and passing in from the left side. The prostate will be felt projecting into the rectum in the male, and above it is the trigone of the bladder flanked by the seminal vesicles; below it is the membranous urethra. In the female the cervix uteri will be felt projecting back in the form of a hard knob. The mucous membrane must be examined for polypi, ulcers, etc.

It must be remembered that hæmorrhoids are not palpable. The presence of scybala or foreign bodies can be determined, and the existence of any tumour, either in the bowel or pressing upon it, can be made out. If the lymphatic glands which lie in the hollow of the sacrum are enlarged, they can be felt.

SECTION IV.—CHEMICAL INVESTIGATION OF THE STOMACH CONTENTS.

The object of this is to test the digestive and motor power of the stomach.

A "**test meal**" is given, and the stomach contents withdrawn, after a stated interval, and examined. The best test meal consists of a lightly-boiled egg or one ounce of mince, 3 ounces of toast, and a quarter of a pint of weak tea, but almost equally good results can be obtained by examining the contents of the stomach after any ordinary light meal which contains sufficient proteid and the nature of which is known. After such a light meal as the above the stomach tube may be passed in two hours; if a heavier meal has been taken it may be necessary to allow the lapse of four or even six hours.

Method of withdrawing contents. — Pass the stomach tube as described at p. 51, but have it connected with a glass or vulcanite funnel. The patient should sit on a chair placed on a square of waterproof sheeting. He should be wrapped in a mackintosh and sit with his legs apart, so that anything spilt on the mackintosh may run into a pail placed between them.

When the tube has reached the stomach ask the patient to retch or strain, the end of the funnel being depressed at the same time. In this way a sample of the contents can usually be obtained and caught in a clean vessel. Should this fail, press over the epigastrium while the patient leans forward and try to drive the stomach contents up into the tube. Should the contents still fail to come away the following procedure, as recommended by Gillespie,* may be tried:—"Fill the funnel with warm water, pinching the india-rubber tube a short distance below with the left hand, so that no air can get down; then relaxing the pressure, let the water fill the tube to some way beyond the piece of glass inserted in the middle of it. Then, when just enough water has been allowed to pass down to fill the tube, lower the funnel, let the water run into the pail, and the contents of the stomach are sucked up by the syphon-action thus induced, and may be caught, when they appear after the water, in a clean basin held for the purpose. If it is done carefully, little or no water enters the stomach, and hence the contents are not diluted."

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* "Modern Gastric Methods," p. 18.

If one still fails, one can run into the stomach a measured quantity of water to start the syphon-action, and deduct the amount used in subsequent calculations, or one may even be obliged to aspirate the contents by means of a rubber ball syringe (such as is used for giving an enema) or a partially exhausted aspirating bottle. As far as possible, however, any method of aspiration should be avoided, as none is free from some risk.

When the stomach contents have been obtained they should be allowed to settle in a tall jar, after which one can proceed to examine them. If necessary, they may be decolorised by shaking them up with animal charcoal. The following points have to be ascertained :—

1. **Are the contents acid?**—Test with litmus paper.

2. **Is the acidity due to free acid or to acid phosphates?**—This may be tested in two ways :—

(*a*) Dip in a piece of Congo red test paper. (Appendix, 8.) Free acids turn this blue; acid phosphates do not.*

(*b*) Take 5 cc. or so of the fluid in a test tube and add to it a pinch of calcium carbonate. If effervescence occurs, free acid is present. Filter. If the filtrate still retains its acidity, then acid phosphates are present.

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* Combined hydrochloric acid, if present in large amount also gives a blue reaction

This test depends upon the fact that free acid is neutralised by calcium carbonate, while acid phosphates are not.

3. **How acid are the contents?**—*i.e.* **total acidity.**—Take 20 cc. of the original fluid (unfiltered), add 280 cc. of distilled water, and shake up thoroughly, so as to break up any particles. Divide into two equal portions of 150 cc. each, and add to each a few drops of phenol-phthalein solution. Place the two portions in separate flasks, and titrate one with decinormal soda solution, keeping the other as a standard. Stop running in the soda whenever the least trace of flesh colour appears in the flask. This can best be appreciated by holding the two flasks alongside each other against a white surface. The process may then be repeated with the other flask, and the results compared.

The result may be stated in one of two ways: (1) Directly, the number of cc. of decinormal soda solution required to neutralise 100 cc. of the stomach contents being taken as the "acidity"—*e.g.* if 10 cc. of the stomach contents be titrated, and 5 cc. of decinormal soda run in before a pink tinge is produced, then 50 cc. of soda would be required for 100 cc. of the fluid, and the acidity is 50. The normal acidity of stomach contents varies from 30 to 70.

(2) One may express the result in terms of HCl. Thus one litre of decinormal soda is required to neutralise 3·65 grms. of HCl. If, therefore, 100 cc. of stomach contents require 50 cc. of soda to neutralise them, then the acidity of the 100 cc. is equal to that

of 0·18 grm. HCl.—that is to say, the acidity is 0·18 per cent. The normal total acidity in terms of HCl. is about 0·2 per cent.*

4. **Is the free acid present mineral** (*i.e.* HCl.), **or organic** (*i.e.* lactic, acetic, butyric), **or both?**

Günzburg's test for free HCl.—Place ten drops of the stomach contents in a porcelain capsule, add an equal quantity of the phloroglucin and vanillin solution (Appendix, 5). Heat gently, taking care to avoid charring. When almost dry complete the evaporation by blowing on the surface of the fluid. If free hydrochloric acid is present a pink colour appears, usually at the periphery of the dried fluid.

The reaction is only given by *free* hydrochloric acid. The combined acid and organic acids do not yield it.

Boas's resorcin reagent (Appendix, 6) may be used similarly. It gives a purplish colour. This method is less sensitive but much more economical than that of Günzburg.

Tests for organic acids.—It is best first to dissolve these out by means of ether. Shake up 10 cc. of the fluid with 50 cc. of ether very thoroughly in a separation funnel or tall cylinder. Pour off the ether. Divide the ethereal extract into two equal portions, and place these in wide beakers. Set one in hot water; allow the other to evaporate slowly at the temperature of the room. In the former all the ether will soon have disappeared. Dissolve the residue in about 5 cc. of water. Take some of Uffelmann's reagent (Appendix, 7) in a test tube, and add to it a few drops of the watery solution. If the blue solution changes to yellow, **lactic acid** is present.

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* The necessity for calculation may be avoided if one remembers that, provided 10 cc. of contents have been taken and that *deci*-normal soda is used for titration, the number of cc. of soda required × 0·365 = HCl. per 1,000 parts. In order to get the percentage of HCl. one has merely to shift the decimal point one place to the left. For example, if 10 cc. of soda solution were required to neutralise 10 cc. of gastric contents, the amount of HCl. present is 3·65 per 1,000 or 0·365 *per cent.*

The residue of the second beaker is used to test for acetic and butyric acids. These, being volatile, would be driven off unless the ether had been evaporated at a low temperature. Dissolve the residue in a little water. Neutralise part with a little carbonate of soda, and add to it some *very dilute* neutral perchloride of iron solution. A claret-red colour indicates the presence of **acetic acid.** To the other part of the solution of the residue add a small fragment of calcium chloride. If oily drops appear on the surface, **butyric acid** is present.

Acetic acid can also be recognised by the odour of vinegar; butyric acid by its characteristically rancid smell.

In carrying out the above tests, it is well to filter the stomach contents first through fine muslin, and to use only the filtrate.

An approximate idea of the proportion of the total acidity which is made up of free HCl., combined HCl., lactic acid, and other organic acids respectively, may be arrived at by a simple method described by Gillespie.* This method depends upon the fact that if a portion of the stomach contents is evaporated to complete dryness at boiling temperature, lactic and combined hydrochloric acids are not volatilised, whereas free HCl. and other organic acids are.

10 cc. of the filtered or unfiltered gastric contents are evaporated in a basin placed on a water bath or floated on a pan of boiling water. The basin is left for at least an hour after its contents seem to be dry. A small quantity of distilled water is then added

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* "Modern Gastric Methods," p. 74.

and a drop tested with phloroglucin vanillin. If free hydrochloric acid is present the evaporation must be repeated, if need be, thrice. Ultimately the residue is stirred up with distilled water, and its acidity estimated by decinormal soda solution. The result gives the amount of acidity due to combined HCl. and lactic acid, or, if a qualitative test has shown absence of the latter, to combined HCl. alone. The presence of acid salts may be ignored. The difference between the acidity after evaporation and that of an equal quantity of the fresh contents gives the amount of acidity contributed by free HCl. and volatile organic acids (acetic and butyric). As the residue left after evaporation is often highly coloured some difficulty may be experienced in determining the exact point at which the end reaction occurs. The best way of overcoming this difficulty is to divide the re-dissolved residue into two parts, and to titrate one only, keeping the other as a standard of comparison.

5. **Are albumoses present?**—Place in a test tube two or three drops of a 10 per cent. solution of sulphate of copper. Invert the test tube so that most of the solution runs out again. As much will adhere to the sides as is required for the test.

Neutralise a little of the filtered stomach contents, and add about 1 in. of the fluid to the copper solution. Then add about an equal amount of caustic soda solution (10 per cent). If albumoses are present, a reddish or pinkish colour is produced (biuret reaction).

The presence of albumoses proves the presence of pepsin in the gastric juice. In the absence of albumoses, **pepsin** can be tested for by its digestive action on egg albumen.* Strips of hard-boiled white of egg are cut with a double-bladed knife and discs punched out by a cork borer. These are preserved in glycerine till required. To each of four test tubes 5 cc. of filtered gastric contents and a disc of egg-white are added. The first tube receives no further addition; to the second add two drops of dilute hydrochloric acid (B.P.); to the third, three grains of pure pepsin; and to the fourth, both acid and pepsin. Keep all the tubes about body temperature. The rate at which the disc dissolves in the different tubes will show which ingredient, if any, is deficient in the gastric juice.

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* It is only necessary to test for pepsin if free HCl. is absent.

Product of test breakfast in health.—If a test breakfast be given to a healthy person and the contents removed one hour afterwards, it will be found that 20 cc. to 40 cc. of fluid are obtained. This is transparent, straw-coloured, of an acidity equal to that of 0·2 per cent. or so HCl.; it contains free hydrochloric but no organic acid. Albumoses are present.

In some functional disorders of the stomach the total acidity of the contents is increased, rising above that of 0·2 per cent. HCl. Sometimes no free HCl. is found. In cases of malignant disease of the stomach the absence of HCl. is of sufficient constancy to be of diagnostic value. The presence of organic acids is an indication of the existence of abnormal fermentative processes in the stomach.

The **absorptive power** of the stomach cannot be satisfactorily tested.

The **motor power** is best tested by means of a test dinner. Seven hours after such a dinner the stomach should contain no food, and its contents should be neutral. If expulsion of the contents has not been active the fluid withdrawn will have an acid reaction, and will show the presence of albumoses. The presence of large lumps or fragments indicates enfeeblement of the churning power.

SECTION V.—EXAMINATION OF THE VOMIT.

1. **Naked-eye characters.**—The general character of the vomit varies greatly, of course, with the nature of the food which has been taken. In *dilatation of the stomach* the vomit is apt to be very copious, sour-smelling, and after standing exhibits a froth on the surface. *Bilious vomit* is yellow or green in colour; *fæcal vomit* presents a very similar appearance, but is distinguished by its fæcal odour and by its neutral or alkaline reaction. The presence of much *mucus* gives to the vomit a viscid consistence. The appearance of the vomit in hæmatemesis varies. If the bleeding be very copious, the vomit may present the appearance of pure blood and may contain clots. Such bleeding may proceed from a gastric ulcer or from varicose œsophageal veins. More commonly the blood is altered in colour by being retained for some time in contact with the gastric juice. Thus it may be blackish in colour or dark brown. The latter appearance is due to the conversion of hæmoglobin into hæmatin. The altered blood gives to the vomit an appearance often compared to that of *coffee grounds* or hare soup. It should be borne in mind that the taking of preparations of iron or red wines may produce a very similar appearance in the vomit. Vomit which contains dark green bile may resemble very closely vomit which contains blood. On diluting with water, however, the green colour of the bile becomes more apparent, while blood remains dark.

2. **Chemical examination.**—The vomit should be filtered through fine muslin. The filtrate can then be examined, if desired, in the manner already described for the stomach contents.

Bile can be detected by Gmelin's test (p. 354).

For the chemical detection of **blood** in the vomit the guaiac test is not satisfactory. It is better to take up some of the brown deposit with a pipette, to place it in a porcelain capsule, and add a pinch of powdered chlorate of potash and a few drops of strong hydrochloric acid. Heat till dissolved. Cool and add a few drops of ferrocyanide of potash solution. A blue colour indicates that blood is present. The reaction is due to the iron contained in the blood pigment. If the patient has been taking iron the test is, of course, inapplicable. In such a case some of the deposit should be digested with caustic potash, filtered, and the solution examined for the spectrum of alkaline hæmatin, or the deposit may be subjected to Teichmann's test (Appendix, 19). To confirm the test, add to the alkaline hæmatin solution a few drops of sulphide of ammonium, which converts it into hæmochromogen. The spectrum of the latter is identified by its possessing two bands, one, narrow and dark, in the yellow between D and E, the other, broader and less dark, at the junction of the yellow and green between the lines E and b (Fig. 73, p. 232).

3. **Microscopic examination** (Fig. 11).—Take up some of the deposit which adheres to the muslin, spread it out on a slide, and examine either directly or in a drop of salt solution.

Various particles derived from the food may be recognised. *Muscle fibres* by their transverse striæ. *Starch granules* by their concentric lines and the fact that a drop of very dilute iodine solution turns them blue. *Elastic fibres* by their double contour and bold curves. *Fatty particles* by their high refractility.

Various **vegetable parasites** may be present. The most important are the **sarcina ventriculi** (a large micrococcus) and the **yeast fungi.** The former can be recognised by their forming small cubical packets of cells resembling miniature bales of wool; the latter consist of round or oval cells in chains or clusters. They are usually about the size of white blood corpuscles.

The addition of a very little dilute iodine solution to the vomited matter may render the detection of sarcinæ more easy. The iodine stains them a deep mahogany brown.

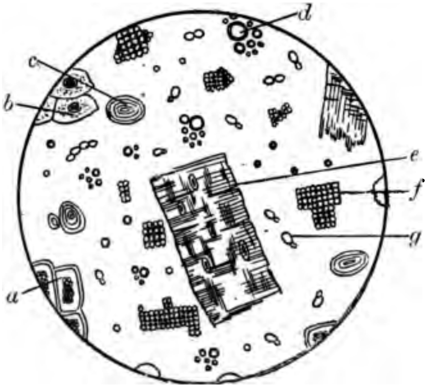

Fig. 11.—Microscopical view of vomited matter.

a, vegetable cells; b, epithelial cells; c, starch granule; d, oil globule; e, muscle fibre; f, sarcina ventriculi; g, torula.

Permanent preparations of these fungi may be made by spreading out some of the deposit in a thin layer on a cover glass and drying over a flame. The best stain for sarcinæ is an extremely dilute—almost transparent—solution of gentian violet. Stain for a minute or two. Bismarck brown also gives good results; stain very briefly. For yeasts use a 2 per cent. solution of methylene blue, and stain for half a minute. Wash in water in both cases, dry between filter papers, and mount in balsam.

SECTION VI.—EXAMINATION OF FÆCES.

1. **Naked eye.**

The following points should be attended to:—

(*a*) Amount of the daily stools;
(*b*) Their colour;
(*c*) Their odour;
(*d*) Their consistence and form;
(*e*) The presence of any abnormal ingredients.

As regards **amount,** it is usually sufficient to state whether the stools are copious or scanty. The average daily amount of fæces in health is 120-180 grms. (about 4 ozs.).

The **colour** of normal fæces is partly due to urobilin, partly to chlorophyll and other pigments. The presence of unaltered bile pigment is always abnormal, and may be due to increased rapidity of peristalsis. *Black* stools may be produced by the administration of iron, bismuth, or manganese. In hæmorrhage high up in the intestine the altered blood makes the stools dark, tarry-looking, and very offensive. The blackness due to blood may be distinguished from that produced by drugs by mixing part of the stool with twice its volume of water and allowing it to stand in a glass jar. If blood be present the water becomes reddish: under other conditions it remains dark or greenish.

Pallor of the stools may be due to an obstruction to the entrance of bile into the intestine, as in jaundice, or to extreme dilution of the stool, as in cholera.

The **odour** of the fæces is due to the presence of indol and skatol. The absence of bile seems to favour putrefaction, hence the stools in jaundice are often very offensive. Cholera stools, on the other hand, contain very little organic matter, and are almost free from odour. In fermentative processes in the intestine the stools may have a sour smell.

The **form** and **consistence** of the stools is of importance. In obstinate constipation the stools may be much drier and harder than normal, and even friable. In all forms of diarrhœa they are more fluid than normal, and may even be watery. Slimy stools are due to the presence of an excess of mucus.

It is important to note whether the stools are formed or fluid. If formed, any abnormality in the shape should be noted. The stools of constipation have often the form of round balls, frequently coated with mucus. In obstruction in the large intestine the stools may be ribbon-like. If ascites is present, the pressure of the fluid on the bowel often leads to flattening of the fæces. The presence of a rectal polypus may produce a groove or furrow along the fæcal mass.

In order to facilitate the detection of **abnormal ingredients**, the stool should be placed on a fine sieve, and a large quantity of water added. The whole is then shaken and stirred up till the soluble parts are all washed away. The residue is then examined.

Gall stones are easily recognised. It is important to note whether they are facetted or not, for, if they are, then the stones are multiple. Particles of undigested food, fruit stones, foreign bodies, concretions —*e.g.* those produced by magnesia—and parasites should all be looked for.

The full consideration of the parasites which may be found in the stools is undertaken later. We would only mention here that one has often to search stools for the head of a tapeworm. The best method of procedure in such a case is to add to the stool a considerable quantity of water containing a little carbolic acid, and to shake the mixture gently for a few moments. It is then allowed to stand for about ten minutes. The parasite sinks to the bottom, the supernatant fluid is poured off, and more water added till the residue is nearly colourless. The parasite will then be readily found. The head is only about as large as that of a large pin, and the neck about as thick as a stout thread.

Special terms are applied in clinical medicine to some particular varieties of stool.

The *bilious* stool is well illustrated in the typical stool of typhoid fever. Its characters are described in the term of "pea soup" stool, usually applied to it.

Watery stools are found in all cases of colliquative diarrhœa, and after the administration of hydragogue cathartics. To the watery stools of cholera the special name of *rice-water* stools is applied. Such a stool is colourless, devoid of odour, alkaline in reaction, and contains a number of small flocculi, consisting of shreds of epithelium and particles of mucus. The name is applied to it from its resemblance to the water in which rice has been boiled. *Purulent*, or pus-containing, stools are found in severe dysentery or intestinal ulceration, or in cases where an abscess has found its way into the intestine. *Slimy* stools are due to the presence of an excess of mucus, and point to an affection of the large bowel. The mucus may envelop the fæcal masses, or may be intimately mixed with them. *Bloody* stools vary in appearance according to the site of the hæmorrhage. If the latter takes place high up, the stools look like tar, as has been already mentioned. In an ordinary intussusception the stools may look like red currant jelly. In those rare cases in which the intussusception occurs in the jejunum the appearance of the material passed per anum has been compared to that of a melted strawberry ice. If the hæmorrhage be from the large intestine, the blood is less intimately mixed with the fæcal matter, and may even be of a bright colour. In hæmorrhage from the rectum or anus it may merely streak the fæcal masses.

In membranous colitis the motions consist almost exclusively of casts of the bowel, which are mainly composed of mucin. The individual casts vary considerably in size, being commonly from one to six inches long, but in exceptional cases attaining a length of a foot or more. They vary in width from narrow strips to tubes more than an inch in diameter; they are sometimes grey and transparent like ordinary mucus, at other times they are more opaque and resemble the casts formed in the air-passages in membranous croup. Sometimes they are stained of a brownish yellow colour from adherent or incorporated fæcal matter; rarely they are red from the presence of blood. They are often very abundant, and may become agglomerated into irregular masses which, when floated on water, can be unravelled into their component parts. As a rule the cast is tubular, but the wall varies much in thickness, and the lumen may be obliterated. If teased out in water one can sometimes separate the cast into membranous layers, between which small particles of fæcal matter may be observed.

These casts, when small, may, on casual observation, be mistaken for segments of tapeworm. On the other hand, cases are recorded where the remains of indigestible substances in the food or firm clots of curdled milk have been regarded as casts.

Chemically they are mainly composed of a mucin body with which small amounts of globulin and other albuminoids are associated. The presence of fibrin in any quantity appears to be very uncommon, if, indeed, it ever occurs.*

When examined under the microscope without any preliminary fixation, the casts resemble ordinary intestinal mucus, and are seen to consist of a hyaline matrix in which great abundance of cylindrical epithelium is embedded. The epithelium is often aggregated in clumps, some of which by their form appear to have been derived from Lieberkuhn's crypts, and it may either be well preserved or shrunken and vacuolated. It is unusual to find many pus-cells in the cast. Bacteria are always present—sometimes in enormous numbers—and traces of fæcal matter, as well as crystals of cholesterin and of triple phosphate, are usually visible.

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* If present, fibrin may be demonstrated by fixing and hardening a small portion of the cast with sublimate and alcohol and, after preparing a section for the microscope, staining it with Ehrlich's triple stain, when the mucin is coloured green, whilst albumins and fibrin appear red.

Sections of hardened specimens show that the wall is composed of successive laminæ.

2. Microscopic examination of fæces.

If the stool is solid, a small particle of it should be picked up with forceps, placed on a slide, and mixed with a little salt solution. If the stool is liquid, a portion of the deposit should be removed with a pipette and placed on the slide without salt solution. A cover glass is put on in either case, and the specimen examined directly. Should it be desirable to use any stain, a little very dilute watery eosin is to be recommended for the purpose.

One may find (1) particles of food—as already described in the vomit; (2) cells—red blood corpuscles, intestinal epithelial cells, pus corpuscles, or leucocytes; (3) crystals — *e.g.* triple phosphates, phosphate of lime, cholesterin, fatty and hæmatoidin crystals. None of these has any pathological significance. (4) Parasites and bacteria may also be found. The latter are considered in Chapter XIV.; the former demand a more detailed description here. The parasites which occur in the intestinal tract include worms and protozoa. The worms belong either to the nematoda or to the flat worms, the latter group containing the cestoda, which are fairly common, and the flukes, which, in Europe at least, are by no means ordinarily found in man.

(A) NEMATODA.

1. Perhaps the commonest of all internal parasites is the small threadworm, **Oxyuris vermicularis,** whose presence is associated with considerable itching about the anus. It inhabits the large intestines, cæcum and vermiform appendix, and specimens can often be seen wriggling about in the recently-passed motions of their host. To the naked eye they look like small white threads, from a half to one centimetre in length. Under the microscope the female may be distinguished by the large uterus filled with ova, and the pointed posterior end, whence its name is derived (Figs. 12, 13, 18, A).

Fig. 12. — Oxyuris vermicularis. 1, female; 2, male. Nat. size. (*After Payne.*)

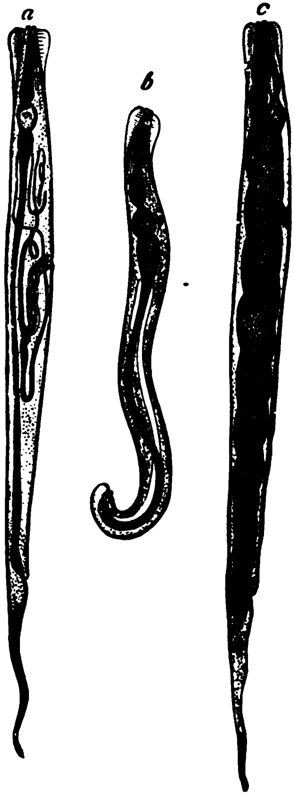

Fig. 13.—Oxyuris vermicularis. *a*, young female; *b*, male; *c*, mature female. Magnified. (*After Payne.*)

Fig. 14.—Ankylostoma duodenale.
a, male; *b*, female. Nat. size. (*After Payne.*)

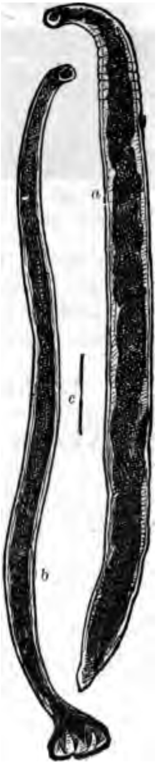

Fig. 15.—Ankylostoma duodenale. *a*, female; *b*, male Magnified. *c*, nat size. (Bristowe.)

2. **Ascaris lumbricoides** has a general resemblance to an earth-worm. It measures, as a rule, from six to eight inches, and sometimes considerably exceeds this length. Not infrequently its presence in children is associated with nervous disorders. The ova, which can occasionally be found in the dejecta, have brownish-yellow granular contents, and in many cases the shell is surrounded by an irregular albuminous sheath (Fig. 18, B).

3. **Ascaris mystax,** a closely-allied worm, is sometimes parasitic inchildren; the infection is got from cats.

4. **Ankylostoma duodenale** is a parasite whose presence is fraught with much greater risk to the host than that of those already mentioned, as it causes profound anæmia by drawing blood from the wall of the bowel. It livesfor the most part in the upper part of the jejunum, and its existence there is rendered probable, when, in an infested district, severe anæmia, otherwise inexplicable, sets in. The diagnosis is clinched by the discovery of ova in the motions. They exhibit a segmented yolk, enclosed in a thin shell, and are sufficiently numerous to be readily detected. The adult worm, which is rarely seen before

therapeutic agents have been employed, is about half an inch long, and the mouth is provided with four claw-like teeth. The male is distinguished by its lobed caudal bursa (Figs. 14, 15).

5. **Trichocephalus dispar** is, perhaps, a commoner parasite than might be suspected; its presence does not seem to cause any very serious inconvenience. The length of the worm is under two inches, its colour is white, the anterior portion is much narrower than the posterior, and is buried in the mucosa of the cæcum. The ova are very characteristic, and when present are readily recognised (Figs. 16, 18, c).

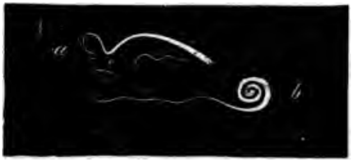

Fig. 16.—Trichocephalus dispar.
a, female; *b*, male. Nat. size (*After Payne.*)

6. **Trichina spiralis** occurs in the alimentary canal in the sexually mature state. The striped muscles are the habitat of the embryonic form, and when much affected they may become shortened. This is well seen in the biceps, where the contraction induces a very typical flexion of the forearm. The adult male measures 1·5 mm. in length; the female, which is viviparous, is about twice as long.

(B) CESTODA.

Many different kinds of tapeworm have been found as parasites in man, but those of most importance are Tænia solium, T. mediocanellata, and T. echinococcus. Tænia cucumerina is also occasionally found in children. Besides its occurrence in the fully developed state, T. solium may be present in the tissues, in the form of a cysticercus; T. mediocanellata is almost never found in this condition in man; whilst T. echinococcus always occurs in the cystic stage, and has never been found in the mature condition in the human intestinal tract (Fig. 17).

The presence of an adult tapeworm in the bowel is generally revealed by the passage of ripe proglottides in the stools, and after the administration of anthelmintics the head may be detected by the methods previously described.

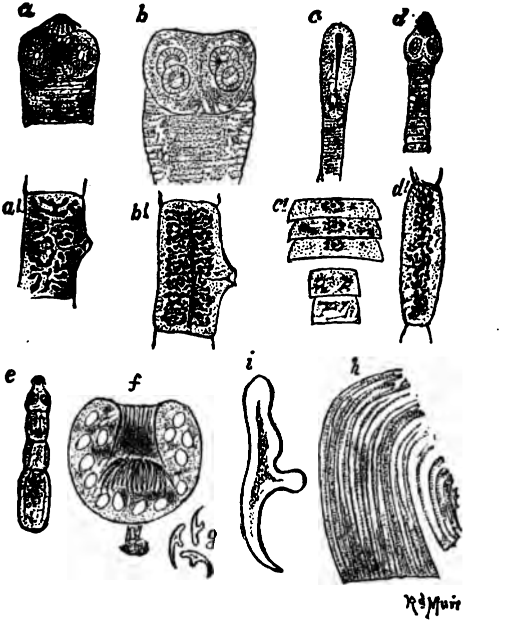

Fig. 17.—Cestoda.

a, head of Tænia solium, × 10; *a′*, mature segment of do., nat. size; *b*, head of T. saginata, × 10; *b′*, mature segment of do., nat. size; *c*, head of Bothriocephalus latus, × 10; *c′* mature segments of do., nat. size; *d*, head of T. cucumerina, × 10; *d′*, mature segments of do., nat. size; *e*, T. echinococcus, × 10; *f*, hydatid scolex (invaginated), × 250; *g*, hydatid hooklets, × 250; *h*, hydatid membrane (ectocyst), × 250; *i*, hooklet from cysticercus, × 25

1. **Tænia solium** (Figs. 17, *a*, *a*′, 18, D). The mature worm measures 2 or 3 yards in length; a ripe proglottis is about 10 mm. long, and 6 mm. broad, with the sexual opening placed laterally; the uterus is coarsely branched. The head, whose size is about the same as the head of a large ordinary pin, has four suckers, often pigmented, and a small rostellum, with a ring of 20 to 30 hooklets. The ova, which are nearly spherical, are readily recognised by their thick shell, with radiating striations. Inside the ovum, when mature, the six hooklets of the embryo may be visible.

The cysticercus varies in size according to its situation, but never attains anything approaching the magnitude of an echinococcus cyst. The vesicle contains one head only, whence the adult worm is developed.

2. **T. mediocanellata (saginata)** is larger than T. solium, and attains a length of 5 to 9 yards. The ripe proglottides measure 16 mm. in length, by 5 mm. in breadth, but immature segments are broader than they are long. Very often they exhibit movements after they have been detached from the strobilus and have passed from the bowel. The sexual opening is lateral, and the uterus is finely ramified, with frequent dichotomous divisions of the primary branches. The head is rather square in outline, and is larger than that of T. solium. It has four suckers, but is devoid of hooklets. The ova closely resemble those of T. solium, but are slightly longer in proportion to their breadth (Figs. 17, *b*, *b*′, 18, E).

3. **T. echinococcus.**—The adult worm, which consists of a head and three segments, and whose length is only four or five millimetres, need not be fully described, since it is not found in man. The *cystic stage* is very important, as it gives rise to serious disease in many of the viscera, and especially

in the liver.* The cysts of this tænia are not simple, but produce, from their inner surface, one or two generations of secondary vesicles, on which the brood-capsules, containing the cestode heads, are formed. During the period in which this process is going on the primary vesicle dilates to accommodate its increasing contents, and may eventually reach the size of a cocoa-nut. The vesicles may rupture spontaneously and their contents may escape by the lungs, by the bowel, or by the urinary passages, or specimens may be obtained by aspiration, or after surgical interference. In a case of suspected hydatid disease one may require to found the diagnosis either on the chemical nature of the fluid withdrawn, or on the recognition of hooklets or scolices, or on the appearance of the ectocyst, portions of which are sometimes discharged, especially when the cyst has opened into the lungs and bronchi (Fig. 17, *e*, *e′*, *f*, *g*, *h*).

The *fluid* is clear, alkaline, devoid of albumen, and contains abundance of sodium chloride and traces of glucose. Its density is low, being generally under 1·010. The appearance of echinococcus *hooklets* is shown in Fig. 17, *g*, *i*. The *scolex*, if it is obtained in a perfect condition, is about 1 to 1½ mm. in diameter, and a number of them often spring in a group from one brood-capsule. They have four suckers and a crown of hooklets. Portions of the *ectocyst* appear as whitish-yellow shreds, which can be recognised under the microscope (× 250 diam.) by their lamination, and by the pectinate markings on the laminæ (Fig. 17, *h*).

4. **T. cucumerina** and **Bothriocephalus latus**, though rare in Britain, are common in some parts of Europe. The appearance of the head, and a

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* The so-called "hydatid thrill" is described elsewhere (p. 26). It may occur where there are no daughter cysts, and it may be absent when daughter cysts occur.

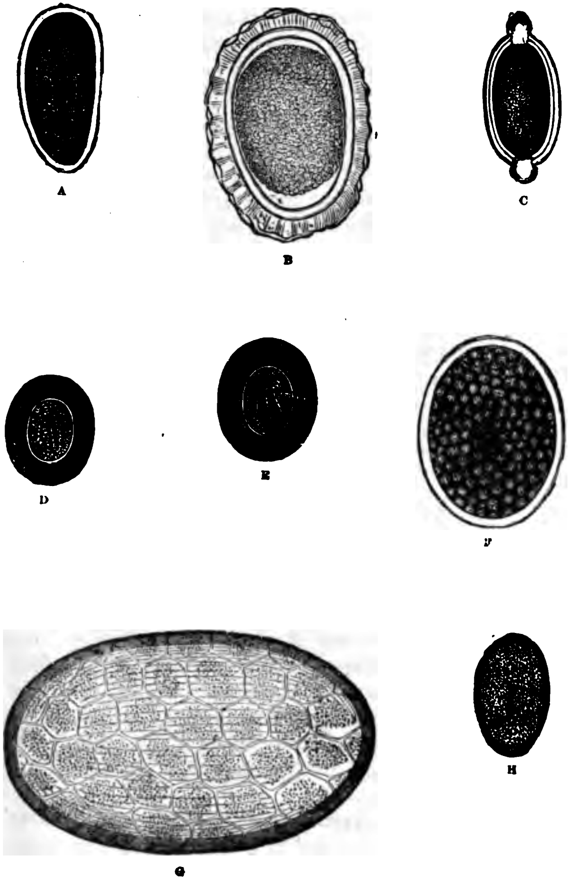

Fig. 18.—Ova of Entozoa, × 350. (*After Heller.*)

A, oxyuris vermicularis; B, ascaris lumbricoides; C, trichocephalus dispar; D, tænia solium; E, tænia mediocanellata; F, bothriocephalus latus; G, distōma hepaticum; H, distōma lanceolatum.

mature segment of each is shown in Fig. 17, *d*, *d'*, *c*, *c'*, and the ovum in Fig. 18, F.

(C) TREMATODA (Fig. 18).

1. **Distōma hepaticum** (Fig. 18, G) is rather rare as a human parasite. When it does occur, the ova may be found in the fæces, and are recognised by their brown colour, and by the presence of an operculum at one pole. The adult fluke is leaf-shaped, and measures about 25 mm. by 12 mm.

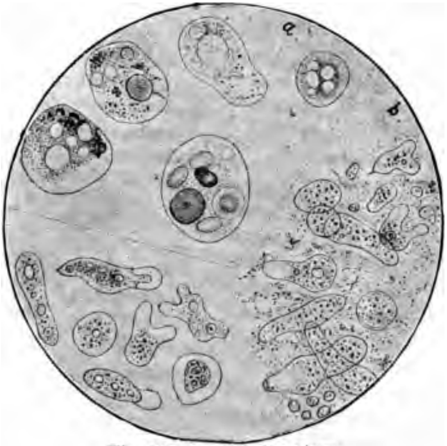

Fig. 19.—Amœbic dysentery.

a amœba dysenteriæ fixed and stained (*Councilman*); *b*, amœba dysenteriæ in stools (*after Lösch*, Virchow's "Archiv," Bd. 65).

2. **Distōma lanceolatum** (Fig. 18, H) is considerably smaller, and is narrower in proportion to its length. The ova are similar to those of D. hepaticum, but smaller.

(D) PROTOZOA.

A number of protozoa, including members both of the Rhizopoda and Infusoria, have been found in the fæces. The only one which is of undoubted clinical importance is the **Amœba dysenteriæ,** which is present in a great proportion of cases of tropical dysentery, and also in tropical abscess of the liver (Fig. 19).

When in a state of activity they vary in form, and throw out pseudopodia, which are thick and few in number; usually only one or two are present. The division of the two plasma layers is indistinct and the ectoplasm is small in amount. At rest they become spherical, measure on an average from 12 to 26μ in diameter, and generally exhibit a nucleus. The resting condition is that in which they are usually found in the fæces, when they are recognised by their high refractility and their greenish tint. For careful examination, Lafleur* recommends that a portion of the dejecta should be hardened in Müller's fluid, and subsequently cut and stained like sections of tissues; or cover glasses, smeared with a thin film of the fæcal material, can be prepared. The stain which he prefers is methylene blue. The amœba is most easily detected in the little masses of mucus which occur in the stool, and attention should always be carefully directed to these. When the motion is pultaceous the addition of warm solution of sodium chloride will be found to facilitate the preparation of a suitable film, in which the organisms may be seen moving about if care be taken to maintain the slide at the temperature of the body. If the motion is a formed one the mucus adhering to its surface should be exclusively examined. In addition to amœbæ dysenteric stools invariably contain bacteria, amongst which streptococci and the bacillus coli communis seem to preponderate.

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* Allbutt's "System of Medicine," vol. ii., p. 755.

CHAPTER IV.

CIRCULATORY SYSTEM.

SECTION I.—ANATOMY.

THIS system is composed of two main elements—the heart and the blood-vessels—and these are for the most part dealt with separately, although, when the chest is exposed for the examination of the heart, the vessels in the thorax and at the root of the neck are more conveniently examined along with it. (*See* Plate I.)

The heart lies obliquely in the thorax, being inclined from above downwards, forwards, and to the left. Two-thirds of it lie to the left of the middle line. The part which reaches highest in the thorax is the *left auricular appendix*, which in the cadaver extends as far up as the second left costal cartilage. During life it is usually opposite the second interspace or lower border of the second cartilage, as the diaphragm then occupies a lower level. The greater portion, however, of the *left auricle* lies posteriorly, and constitutes the hindmost cavity of the heart.

The *right auricle* is the chamber that lies most to the right. It extends somewhat beyond the right margin of the sternum, and its border may be traced by a curved line joining the third and seventh right chondro-sternal articulations, and reaching about one inch * to the right of the sternum.

The *right ventricle* occupies the great portion of the front of the heart. Its inferior margin extends from the seventh right chondro-sternal articulation to the apex, and constitutes the lower border of the heart.

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* Luschka.

The *left ventricle* only appears in front as a narrow strip, scarcely half an inch broad, and its outline completes that of the heart on the left, where its border forms a curved line, ascending from the apex to the lower margin of the second left interspace at a point just internal to the parasternal line.

The topographical anatomy of the valves of the heart and of the great vessels will be discussed in connection with auscultation, as it is in this department that a knowledge of their situation is most necessary (pp. 145–172).

The most important organs which come into relation with the heart are the lungs on either side, the liver below, and the great vessels above. A small portion of the anterior surface is only separated from the thoracic wall by the anterior mediastinum, whilst behind, the heart is in relation with the structures that occupy the posterior mediastinum.

That portion of the anterior aspect of the chest which overlies the heart is known as the **præcordial region.**

It is often necessary to define the exact situation of a point on the front of the thorax, and certain landmarks, some natural and some artificial, are commonly made use of for this purpose.

The ribs and interspaces on either side form convenient horizontal landmarks. In order to count them, one must feel for the ridge which marks the junction of the manubrium with the body of the sternum, known as the angle of Louis.* When this has been found, by running the finger outwards it reaches the second costal cartilage, which articulates with the sternum at this level. It is then easy to reckon upwards or downwards to the other ribs. The

* Angulus Ludovici.

determination of the first rib directly is neither so easy nor so certain, since it is overlapped by the clavicle.

In order to define the distance of any given point from the mesial sagittal plane of the body, a series of vertical lines is imagined to be drawn on the chest. These are the **mid-sternal** and **lateral sternal lines,** drawn down the middle and either border of the sternum; the **mammary line,** best defined, since the situation of the nipple is inconstant,* as the vertical line dropped from the centre of the clavicle, or, what amounts to the same thing, the line midway between the middle of the suprasternal notch and the tip of the acromion; the **parasternal line** midway between the lateral sternal and mammary lines; the **anterior, mid, and posterior axillary lines** descending from the anterior border, the centre, and the posterior border respectively, of the axilla; and the **scapular line,** which is defined as the vertical line drawn through the angle of the scapula.

The methods commonly employed in the examination of the heart are inspection, palpation, percussion, and auscultation. These will be taken up consecutively, although in practice inspection and palpation are often advantageously combined.

SECTION II.—INSPECTION.

Inspection determines :—

A) Form—

1. Of præcordia $\begin{cases}\text{bulging.}\\\text{flattening.}\end{cases}$
2. Of surrounding parts (especially bulging).

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* The ordinary situation of the nipple in an adult male of average development may be placed in the fourth intercostal space, four inches from the mid-sternal line.

(B) Movements—

1. In præcordial region
   - apex beat.
   - diffuse pulsation.
   - pulsation at base of heart.
   - local indrawing.
2. Outside præcordial region
   - Pulsations at root of neck.
   - ,, in thorax.
   - ,, in epigastrium.

(C) Dilated veins and venules.

For inspection of the chest, the patient should be stripped to the waist, and set in a good light, either sitting up or lying on his back. The observer should directly face him, but must be careful not to obstruct the light. In some cases the observer may with advantage take up a position at the top of the bed, and lower his head until he looks along the chest tangentially. By this manœuvre he will be able to study various pulsations with great facility.

The following points must then be systematically noted:—

1. The shape of the præcordia;
2. Pulsations in the præcordial region;
3. Bulging or pulsation outside the præcordia, either at the root of the neck, or the front of the chest, or the epigastrium (scrobiculus cordis);
4. The presence or absence of distended veins on the chest wall or in the neck.

1. The shape of the præcordia.

In health the chest is bilaterally symmetrical, and there is no greater prominence on the left side than on the corresponding area of the right. When the right pectoral muscles are exceptionally well developed, the præcordia may be less prominent than the right side of the chest.

In cases where the **præcordial area is prominent,** it must be remembered that other conditions than disease of the heart may have caused the projection, whilst it is equally to be observed that serious disease of the heart is comparatively seldom accompanied by bulging of the præcordia unless it had already manifested itself when the patient was young and the bones were incompletely ossified.

Should prominence be observed, note whether the ribs are involved, or whether the intercostal spaces alone bulge. The latter condition occurs in pericarditis with effusion. Prominence of the præcordia may be due to disease in the framework of the thorax, such as scoliosis, parietal tumour, or abscess, or to a diseased condition of the thoracic contents, such as cancer of the lung or effusion into the pleural cavity, mediastinal tumour, fluid in the pericardium, enlargement of the heart, especially if it occur in early life, and aneurysm behind or above the heart.

Flattening of the præcordia may be congenital; it may mark the former occurrence of pericarditis; it may be due to retraction of the lung; and in some instances, particularly in certain trades, it may be the result of pressure.

2. **Pulsations in the præcordial region.**

Besides the movement of respiration, which affects the præcordia with the rest of the chest, an impulse which occurs three or four times to each respiration is generally seen in the lowest and leftmost part of that region.

This pulsation is called the **apex beat** of the heart, and in health usually exhibits the following characters :—

Firstly.—It is found in the fifth left intercostal space.

Secondly.—It is limited to an area less than an inch in breadth, and is only visible in one interspace.

Thirdly.—It is situated outside the left parasternal line, and inside the left mammary line; and

Fourthly.—It is due to the impact on the chest wall of the apical segment of the heart, and for clinical purposes the actual apex of the heart may be assumed to be situated at the lowest and leftmost part of the above area of pulsation, although it may really be slightly lower down and farther out, under cover of a rib.

The apex beat may be abnormal in **force,** in **position,** or in **extent.** Even in perfect health, if the chest is well clothed, and the apex lies behind a rib, it may be quite invisible. *Disappearance,* therefore, of the apex beat is not to be regarded as necessarily indicative of disease, though it must not be forgotten that it is in cases of weak action of the heart that it its most frequently absent or diminished in force. When abolished, its place may be taken by a more *diffuse impulse* over the lower part of the præcordial area, in cases where the apex is pushed away from the chest wall by a dilated right ventricle, or when pericardial effusion separates the heart from the front of the thorax. On the other hand, the apex beat may appear to be *more forcible* than usual in cases where the heart's action is excited, where the chest wall is thin, or where the left ventricle is hypertrophied. Such changes are more accurately observed by palpation, and will be discussed under that head.

The **position of the apex beat** may be altered in three classes of cases. The cause may be (*a*) *congenital,* where the heart is reversed so that the apex lies to the right (*dextrocardia*), or where other developmental anomalies are present. The displacement of the apex beat may be due to (*b*) *extrinsic causes,* where the heart is displaced by diseased conditions of surrounding viscera which push or pull it from its usual site. Instances of this are found in pleurisy with effusion, in abdominal tumours, and in retraction of a lung.

Where the heart is pushed over to the right by a left pneumothorax, or pleuritic effusion, the pulsation which is conspicuous to the righthand side of the sternum is not that of the apex, which is usually lying somewhere behind the bone, but is due to pulsation of the right ventricle and auricle.

Thirdly, the displacement may result from (*c*) *disease of the heart or pericardium.* The apex beat is displaced *mostly outwards* when the heart is dilated; *downwards and outwards* when the left ventricle is hypertrophied; and often *upwards* when fluid is present in the pericardial sac.

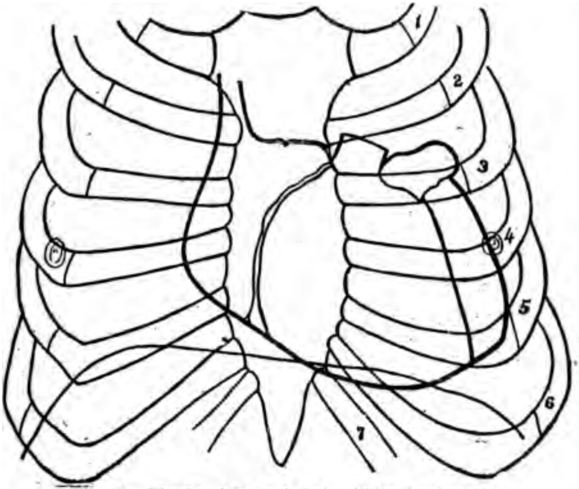

Fig. 20.—The heart in an infant. (*After Symington.*)

In addition to these causes, it should be remembered that the position of the apex beat varies considerably with the patient's age; *in children* it is usually as high as the fourth interspace* (Fig. 20); *in the aged* it descends as low as the sixth.

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* Even in young children, although the apex beat is seen in the fourth interspace, the actual apex lies lower down—usually behind the fifth rib.

In certain cases the apex beat is replaced by an *indrawing* of the same area during cardiac systole. This, when it is marked in degree, and when it appears over an extensive area of the lower segment of the præcordia, suggests pericardial adhesion. When only slightly visible and limited to the apex, it is of no great consequence.

The **extent of the apex beat** is increased in cases where the heart is hypertrophied. This condition must not be confused with diffuse pulsation over the præcordia. *Doubling of the apex beat* sometimes occurs, and may be due to various causes, of which the most important are alternate systole of the right and left heart, and hemi-systole.

The consideration of **other pulsations** which may be visible in the præcordial region must next be undertaken. Allusion has already been made to the diffuse pulsation which occurs when the right ventricle is dilated and hypertrophied, and which in these circumstances is visible over the lower part of the area in several of the intercostal spaces, and nearer the middle line than the normal apex beat.

Pulsation is also seen at times in the second left intercostal space. It may arise either in the pulmonary artery, which lies half under cover of the left side of the sternum, and half under the inner end of this interspace, or in the left auricular appendix. In the former case, it follows the apex beat, and marks the closure of the pulmonary valves; in the latter case, which is not common, it immediately precedes the apex beat.

In chests which have thin parietes, and especially when, in addition, the left lung is retracted from phthisis or other disease, pulsation of a diffuse nature can be observed over most of the interspaces of the præcordial region, as well as at the apex. In these cases the apex beat still causes a limited area of the chest wall between the left parasternal and mammary lines to bulge forward with each beat of the heart, whilst the diffuse pulsation which is caused by the systole of the right ventricle is associated with indrawing of some portion of the intercostal spaces. This retraction, accompanied by a normal apex beat, and not confined to the lower præcordial segment, must not be confused with the systolic indrawing already described as characteristic of pericardial adhesion.

3. **Pulsations outside the præcordia.**

In addition to the pulsations already described, movements should be looked for at the root of the neck, the front of the chest, and the epigastrium.

At the root of the neck, pulsation may occur either in the episternal notch or external to the sternomastoid.

In the episternal notch, the pulsation is usually systolic in time, and when well marked is generally an indication of dilatation or aneurysm of the transverse portion of the arch of the aorta. Less commonly it is due to a thyroidea ima artery of considerable size, or to an abnormal origin of the right subclavian from a point to the left of the middle line. Palpation generally enables these conditions to be discriminated. Pulsation here and in the carotids is not uncommonly seen in cases of chlorosis and in other forms of anæmia.

Outside the sternomastoid various pulsations may be observed. These may be either arterial or venous. The carotids pulsate visibly on exertion; from mental excitement; in diseases which cause excitement of the circulatory system, such as exophthalmic goitre; in cases of hypertrophy of the left ventricle, especially when associated with aortic incompetence; and in aneurysm of the artery.

The jugular veins may exhibit undulation or pulsation. This is usually caused by back pressure of blood on the right side of the heart. It will be discussed under the venous pulse (p. 194).

In the thorax, besides the pulsations referred to as occurring in the præcordial region, a *diastolic pulsation* may occasionally be observed in the second right intercostal space, and results from the closure of the aortic valves. An important source of pulsation in unusual parts of the thorax is *aneurysm of the aorta.* Such aneurysmal pulsations always manifest themselves at first above the level of the fourth rib, though at a later period they may affect a very considerable portion of the chest wall. The position of the impulse varies according to the part of the aorta which is diseased. If the *ascending aorta* is affected, the pulsation is chiefly to the right of the sternum, whilst the *transverse aorta* gives rise to pulsation under the manubrium sterni, and the *descending aorta* still more to the left. Aneurysm of the *innominate* may project far into the neck. The time of this pulsation is systolic, following immediately on the apex beat, and it may be observed to be expansile in character. The pulsation will, of course, be much earlier manifest when the vessel lies behind soft parts than when it is covered by bone.

Pulsating empyema may be present; it generally occupies the præcordial area from which the heart is more or less displaced, and malignant tumours with a large blood supply may also give rise to pulsation in the part of the chest wall that overlies them.

Dr. J. F. H. Broadbent has pointed out that marked *systolic retraction* of some of the lower ribs on the lateral and posterior aspects of the thorax may occur as a result of extensive pericarditic adhesion, involving not only the central tendon, but also the muscular part of the diaphragm on the one hand and the interior wall of the thorax on the other. It usually occurs on the left side.

In the epigastrium there may be several kinds of pulsation. The first thing to be determined as whether it is *strictly systolic*, coinciding exactly with the apex beat, or whether the pulsation is *slightly delayed*, so as to appear just after the apex beat has occurred.

In the former case the pulsation is caused by a dilated and hypertrophied right ventricle, which either conveys its impact directly to the parietes, or does so indirectly by exercising a thrust upon the liver, or else it is due to the apex beat of a heart displaced to the right by some diseased condition, of which the most important are left-sided pleurisy and pneumothorax.

In the case of delayed pulsation, the cause may be arterial. The existence of an aneurysm of the abdominal aorta would produce such an effect. More commonly, however, the condition is simply neurotic; whilst, in other instances, the pulsation of a normal abdominal aorta is conveyed to the surface either by the liver or by an abdominal tumour, such as pyloric cancer, which lies in front of it. (*See also* p. 56.)

In cases of regurgitation from the right heart, pulsation also occurs just after the apex beat, and is due to a distensile pulsation of the liver itself from the back flow of blood into the hepatic veins. It should be observed, however, that distensile pulsation of the liver is by no means a common condition, and does not occur in all cases of tricuspid regurgitation.

It is comparatively rare to observe a *systolic depression of the epigastrium.* If well marked, it would probably indicate adhesions resulting from an old pericarditis.

In order to observe with greater facility the characters and time relations of these various pulsations, one can employ small flags, made as light as possible and attached to the various areas of the chest wall. To determine whether a pulsation is expansile, place one of these flags on either side of the tumour. If it be expansile their free extremities will recede from each other as the tumour fills. If it is desired to time the occurrence of two pulsations, after fixing a flag on the point where each occurs, one may take up a position in which they are as nearly in line as possible. It is then quite easy to determine which of them begins to move first.

Flags can readily be improvised by taking a piece of straw or a bristle about three inches long, fixing a fragment of gummed paper to one end and surrounding the other with a pellet of modeller's wax or stiff ointment which will adhere with sufficient tenacity to the skin. Other more primitive methods may also be used, such as passing a pin through a piece of adhesive plaster, with the head to the sticky side, and fixing it on the chest, or affixing little cones of cotton wool to the points in question by means of vaseline.

4. **Conspicuous veins.**

The veins of the thoracic wall may be unduly conspicuous. This occurs (*a*) when the patient's skin is unusually transparent; (*b*) when the patient has been undergoing considerable exertion, especially when the effort is of such a kind (*e.g.* playing a wind instrument) as to throw a strain on the respiratory system; (*c*) when intrathoracic tumours impede the return of blood to the heart; (*d*) when the action of the right side of the heart is laboured; (*e*) when, in consequence of portal obstruction or of blockage of the inferior caval system, the blood returning from the abdominal viscera or lower limbs is forced to find its way through collateral channels.

In a number of instances, where the right side of the heart is slightly overworked, a belt of dilated capillaries appears along the line of attachment of the diaphragm.

SECTION III.—PALPATION.

Palpation determines* :—

(A) Form of præcordia, etc. [confirms or modifies results of inspection.]

(B) Movements.

- (*a*) Apex beat { position / character.
- (*b*) Other præcordial pulsations.
- (*c*) Pulsations outside præcordia { heaving. / expansile.

(C) Vibrations—

- (*a*) Originating within the heart and bloodvessels. (Thrills.)
- (*b*) Originating exocardially. (Friction.)

By palpation the observer not only confirms the facts determined by inspection and adds to their precision, but is also able to detect movements and vibrations which are too slight to be noted by the eye alone. For palpation the patient should be placed in an attitude which he finds easy to maintain, since the exertion which a constrained position demands is certain to increase the observer's difficulties. If the patient is lying down, care must be taken to keep him on his back. By turning to his left side he will produce a very material alteration in the position of the apex beat, which is thereby displaced outwards towards the axilla; whilst if he lie on his right side the apex of the heart may recede from the chest wall, and an impulse, which in the more favourable dorsal attitude would be easily felt, may entirely disappear.

The position of the observer is almost as important as that of the patient. For the examination of the præcordia he should stand or sit at the top of the bed, on the right-hand side. He should then place his right hand, which must be thoroughly warm, on the patient's chest, so that the palm lies over the base of the heart, whilst the fingers are directed towards its apex. To begin with, the whole palm of the hand should be in contact with the chest wall, and care must be taken not to dig the finger tips into the intercostal spaces, as this causes discomfort, and may thereby interfere with the subsequent observations.

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* The study of the pulse, which also belongs to this department, is for convenience placed by itself in another section.

When pulsation is detected over any part of the region under examination, its exact localisation is best determined by the pulp of the fingers.

The first pulsation to attract attention is that due to the **apex beat.** Not unfrequently the fingers will determine that this is really farther from the middle line than inspection would have led one to suppose. In such a case that point is to be taken as the cardiac apex which is the leftmost and lowest where the finger is distinctly forced up with each beat of the heart. The sensation of a thrust from below raising the finger is important, because in not a few cases where the heart is acting forcibly it communicates some vibration to portions of the chest wall considerably beyond those which actually lie above it.

The observer, having thus determined the site of the apex beat, must study its **extent** and **character.** As has been previously stated (p. 111) it lies in health well outside the left parasternal line, but never beyond the left mammary line, is as a rule confined to one interspace, and seldom can be seen over an area of more than one inch in diameter. These points will now be carefully examined by palpation, and any deviation from them noted. In addition, however, an estimate must be made of the *energy with which the heart is acting*, and the apex beat may be found to differ from the normal—and this can only be recognised by continued practice at the bedside—in possessing a "*heaving*" *character* in cases where the left ventricle is hypertrophied, a *sharp slapping impact* where there is irritability of the heart, or, on the other hand, a *feeble or almost imperceptible tap* when the heart is fatty, or exhausted towards the end of an acute fever.

When the pulsation of the apex of the heart is so feeble as to be imperceptible when the patient is lying down, it often becomes quite distinct if he sits up, and still more if he leans forward. If, however, these postures are uncomfortable for a patient who is seriously ill, it is better to forego such advantages as they afford than to fatigue one whose strength is already taxed to the utmost. The chief causes of **impalpable apex beat** are (*a*) a thick chest wall, (*b*) a feeble heart, and (*c*) emphysema of the lungs.

When analysed, the varying characters of the beat will be found, after due allowance has been made for the thickness of the chest wall and intervening lung, to depend upon the force with which the palpating finger is driven upwards, and upon the celerity and amplitude of the movement of the cardiac apex as it approaches the front of the thorax at each ventricular systole. *A shock* or "*jog*" is sometimes felt at the apex in consequence of sharp closure of the pulmonary and aortic cusps.

In addition to pulsation, vibrations may sometimes be observed at or near the cardiac apex. Such vibrations are termed **thrills.**

The time of their occurrence in relation to the apex beat must be determined. When they commence with the apex beat and continue during the period of ventricular contraction, they are termed **systolic**; if they are felt whilst the ventricles are relaxed, they are termed **diastolic**; if they occur near the close of diastole, when, though the ventricles are still relaxed, the auricles have entered upon systole, and run up to the apex beat, they are termed **presystolic.**

These thrills may be due either to valvular disease, to pericardial friction, or to friction resulting from pleurisy over that part of the left lung which lies in front of the heart. The thrills due to valvular disease will exhibit a more definite relation to the apex beat, both in point of time and situation of maximum intensity, than those whose origin is exocardial. A systolic thrill, best felt at the apex, may indicate mitral regurgitation, though in some cases the thrill of aortic obstruction is very distinctly felt in this area. A diastolic, and still more a presystolic thrill, when it is best felt at or just internal to the apex beat, is so characteristic of mitral obstruction that, if it is clearly present, one may with a high degree of probability assume that lesion to exist, even in the absence of all other signs and symptoms.

Pericardial or pleural thrills will be readily recognised as such when the patient is auscultated (*see* p. 171). They are generally to and fro in character, and are always audible as well as palpable.* Pulsation and thrill may be detected over the right side of the heart when its chambers are dilated and hypertrophied, or when its valves are diseased.

Over the second left interspace **pulsation of the pulmonary artery,** sometimes systolic, sometimes diastolic (*see* p. 114), and of the left auricle, always presystolic, must be sought for; and a thrill may not rarely be detected in the pulmonary artery in certain diseases, especially in exophthalmic goitre (*see* further under Auscultation, p. 166).

Over the aorta, where it approaches the front of the thorax near the sternum in the second right interspace and behind the second right costal cartilage, pulsations

* Sansom, "Diagnosis of Diseases of the Heart," p. 128.

or thrills may also be detected, whilst in cases of aneurysm of the root of the vessel or of the ascending part of its arch, a characteristic expansile pulsation can sometimes, though by no means always, be observed. In many cases a diastolic shock of considerable strength can be felt over the aneurysmal sac. The time of occurrence of all these phenomena must be given with reference to the apex beat.

At the root of the neck palpation will frequently enable one to identify a pulsating vessel, and so clear up a doubtful diagnosis. When pulsation occurs in the episternal notch one should, if possible, try to push the finger below the pulsating vessel. By so doing one may be saved the inconvenience of diagnosing an aortic aneurysm when the patient has merely an abnormal origin of his right subclavian.

By pressing the finger firmly down from the episternal notch behind the upper part of the sternum—due care being exercised not to cause the patient too much discomfort—*a commencing dilatation* of the transverse arch of the aorta may be identified in time to allow of effectual treatment being carried out, since in health the aorta lies at so low a level that its pulsation can scarcely be detected by this manœuvre.

Another method of discovering an early dilatation of the aorta, when the under-side of the arch is the part involved, has been described by Surgeon-Major Oliver and others under the name of **tracheal tugging.** The explanation of this phenomenon depends upon the fact that in passing from the trachea to the lung the left bronchus lies just below the arch of the aorta, and consequently each time that the aorta is distended the aneurysmal dilatation pushes the bronchus downwards before it, and the latter drags in turn upon the trachea, causing it to descend at each beat of the heart. By standing behind the patient and pressing the cricoid lightly upwards with the finger-tips of both hands, whilst the patient keeps his mouth closed and elevates his chin, the downward tug can in many cases be detected with great facility. The phenomenon can also be observed when the finger-tips are applied to the cornua of the hyoid; and the patient is put to less discomfort when this method is adopted.

Pulsations and thrills may be observed in the carotids, and must be fully investigated by palpation. Occasionally a thrill may be felt in the supra-clavicular fossa, where the subclavian artery crosses the apex of the lung. It may indicate a constriction of the vessel resulting from pleuritic adhesion, or disease of the lung itself.

In the epigastrium the fingers should be pressed gently but firmly upwards under the left costal margin when pulsation of the right ventricle is suspected. By this means it may be readily differentiated from pulsation of the liver.

When the **liver exhibits expansile pulsation,** owing to backward pressure in the veins due to tricuspid incompetence, the whole organ will be found to be affected, and in most cases the expansile character of the movement can be distinctly made out. The pulsation is most readily recognised by placing one hand on the fifth and sixth costal cartilages, and the other on the lateral region of the liver in the mid-axillary line. When the right ventricle, by pressing on it during systole, causes epigastric pulsation, the movement can rarely be detected, except in a part of the liver. In cases of doubt as to the nature of epigastric pulsation, a change in the patient's posture, particularly if he is made to assume a knee-elbow position, frequently clears up the difficulty.

SECTION IV.—PERCUSSION.

Percussion determines :—

(A) The boundaries of the heart and surrounding viscera.
    1. Deep dulness.
    2. Superficial dulness.

(B) The presence of certain abnormal conditions of the cardiovascular system, *e.g.*
    (*a*) Pericardial effusion.
    (*b*) Aneurysmal dilatations.

Theory.—When a sharp tap is given over any part of the body, the underlying structures either resound to the blow or merely respond with a dull thud like that which a lump of putty would yield under similar conditions. The former bodies are described as resonant, the latter as dull, on percussion. The resonant structures in the body are the air-containing organs and the bones. The latter emit a sound when struck which is totally distinct from that yielded by the viscera which contain air. It is known as the osteal percussion sound, and is typically heard when the skull is lightly tapped. The cause of resonance is that such bodies as possess it are able to vibrate with more or less regularity. In the case of the bones this power of regular vibration is due to the elasticity of the osseous substance; in the case of a hollow viscus, such as the stomach, colon, or small intestine, it depends on the periodic oscillations of the contained air columns, and on the tension of the limiting stomach or bowel wall; in the lung the factors become more complex, for we have to deal with very greatly subdivided air columns, and the septa are under a considerable degree of tension. Since the exact quality of the resonance which is produced is influenced by the amount, disposition, pressure, and subdivision of the included air, and also by the tension of the walls and septa of the viscus, it is evident that each organ will, on percussion, give out a sound which is fairly characteristic. With a comparatively simple air-space such as the stomach presents, the resonance resembles that of a drum, and is therefore described as *tympanitic*; in the lung, on the other hand, the innumerable septa so break up the air-space that the resonance acquires a peculiar and quite distinctive character. There is no other sound which bears any close resemblance to it; hence it is simply known as *normal lung resonance*, and the student must learn to recognise it from frequent practice.

The depth of tissue which is thrown into vibration by the percussion stroke depends, when other things are equal, on the force of the blow. When the chest wall over two regions of the lung is percussed with equal force, and when in one case a considerable depth of lung tissue underlies the point, whilst in the other only a thin layer of lung intervenes between the surface and a subjacent solid organ, a characteristic difference is observed in the sound produced, just as, when a big drum and a little one are struck, they emit perfectly distinct sounds. The difference of sound depends on several factors, the important points to note being that the thick layer of lung and the big drum emit a sound which lasts longer and conveys an impression of greater resonance and lower pitch. The thin layer of lung and the little drum, on the other hand, yield a sound of shorter duration, of less resonance, and of higher pitch. The exact pitch, however, is always somewhat indeterminate, as it is compounded of a mixture of several independent, more or less tone-like, sounds and their overtones. For practical purposes we may describe the resonance of a thick layer of lung as "*full*," and that of a thinner layer as "*emptier*," the words being employed in a general but sufficiently intelligible sense, to describe the complex impression which the observer readily recognises, but which, under the conditions of clinical work, baffles exact analysis.

When a very light blow is delivered the resonance of only a thin layer of the subjacent lung is elicited, and the sound produced is comparatively empty, even although a thick part of the lung lies below the point of percussion. Hence it follows that the presence of a solid organ underneath the lung will only render the resonance "emptier" when it approaches sufficiently near the surface to encroach upon the layer which is being set in vibration. A firmer stroke would throw a thicker layer into vibration, and in this case a solid body advancing from below would sooner reach the vibrating area, and render the resonance emptier. In other words, when the object aimed at is to detect the presence of an organ which lies underneath a thick layer of lung, then the percussion stroke should be firm. If, on the contrary, the object of inquiry is to ascertain the spot where a resonant viscus terminates, more especially if it grows thin, wedgewise, as its border is approached, it is evident that the end in view will be best achieved by very light percussion, since by this procedure the resonance, though comparatively empty, remains of uniform quality until the edge of the organ is reached, when it is replaced by an absolutely dull thud.

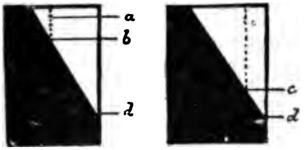

Fig. 21.—Percussion diagram (*see* text)

This is diagramatically represented in the accompanying figure (Fig. 21), where it is easily seen that with firm percussion the resonance begins to grow emptier at *b* and gradually alters thereafter at every point till the emptiness is completed at *d*, whilst with lighter percussion the resonance remains uniform although comparatively empty until *c* is reached, when it rapidly gives place to absolute dulness, and thus the final extinction of resonance is much more readily appreciated.

One other preliminary matter must be referred to. If the tight membrane which is stretched over a drum be covered with a soft solid, the sound which it yields is muffled, and in like manner if a solid organ overlies an air-containing one, the resonance of the latter will be thereby *muffled*, and the more softly the percussion stroke is delivered the more marked will the muffling become. A soft stroke is particularly necessary when the solid organ is thin, or when its border requires to be accurately defined.

Methods of percussion.

When percussion was first introduced, the tap was delivered directly on the patient's skin without the interposition of any substance over the point struck. This method, known as **direct percussion**, is now seldom used except on the clavicles, which in examination of the lungs are lightly tapped by the observer's finger-tip. In order to obtain better resonance, as well as with a view to the patient's comfort, various materials were subsequently interposed between his skin and the percussing finger. A flat plate of bone or ivory, of such a size and shape as to be readily applied and closely adapted to the surface of the chest, is frequently employed, and is called a *pleximeter*. In some instances, when the curvature of the surface renders the application of such an instrument difficult, a short rod of bone or vulcanite, set pillarwise on the region to be percussed, is employed. A very good pillar pleximeter may be improvised by utilising a short piece of good cork, such as is used for closing specimen tubes. It conveys the impact well, and itself emits but little sound when struck. Most physicians, however, prefer to make use of the middle or fore finger of their left hand as a pleximeter, and the preference is due not only to the fact that it can be readily adapted to almost any surface, but also that it often conveys information additional to that obtained by the percussion sound, as it takes cognisance of the different degrees of resistance which the tissues offer to the percussion stroke.

Sometimes a small rubber-tipped hammer, known as a *plessor*, takes the place of the percussing finger, and is occasionally of service; but as a rule the finger should be preferred.

The ordinary method, then, of percussion is conducted in the following manner:—The middle finger of the left hand is placed firmly on the part which is to be percussed, and is adapted to any inequalities of surface, so that no air-space is interposed between it and the skin. The back of its middle phalanx is then struck with the tip of the middle finger of the right hand. The stroke should be delivered from the wrist and finger joints, not from the elbow, and the percussing finger should be so bent that when the blow is delivered its terminal phalanx is at right angles to the metacarpal bones, and strikes the pleximeter perpendicularly. Whenever the blow has been given, the striking finger must be raised, lest it should impair the vibrations it has excited, just as the hammers of a piano fall back from the wires as soon as they have been struck. In cases where the percussion requires to be more firm, several fingers may be used; but it is better, whenever possible, to employ only one percussing finger. In some cases a modification, known as flicking percussion, is useful, and this is particularly valuable in the examination of the abdomen, where the method is more fully described (p. 61).

There are three cardinal rules which should always be remembered when percussion is being carried out. The **first** is that in defining the boundaries between contiguous organs the percussion should invariably be performed from the resonant towards the less resonant. The **second** is that the longer axis of the pleximeter should be parallel to the edge of the organ whose delimitation is being attempted, and the line of percussion should be at right angles to that edge. The **third** is that the pleximeter finger must be kept in firm contact with the chest wall.

It is seldom necessary to deliver more than two or three strokes at any one situation; repeated blows cause much discomfort to a sensitive patient.* The points to be noted on percussion are the *volume* and *pitch* of the resonance elicited, and the sense of *resistance* experienced by the finger.

PERCUSSION OF THE HEART.

The objects which are aimed at in percussion of the heart are twofold: firstly, to ascertain the size and position of the organ as a whole, and secondly, to determine how much of it is uncovered by lung and lies against the chest wall.

Since for the most part the heart is surrounded by resonant lung, but does not lie so deeply as to be out of reach of a firm percussion stroke, one can delimit its extreme boundaries with a considerable degree of accuracy by observing, as one percusses towards the cardiac region, the points at which the lung resonance begins to grow emptier. In two areas this cannot be achieved. At the base of the heart the roots of the great vessels produce a dulling or emptying of the lung resonance which cannot be discriminated from that caused by the heart,* whilst the lower border of the viscus is in relation to non-resonant liver, which yields on percussion the same dull thud that the heart itself does.

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* Students who lack expertness in percussion are advised to gain the necessary skill on a table or some other inanimate object of *varying resonance* rather than on hospital patients.

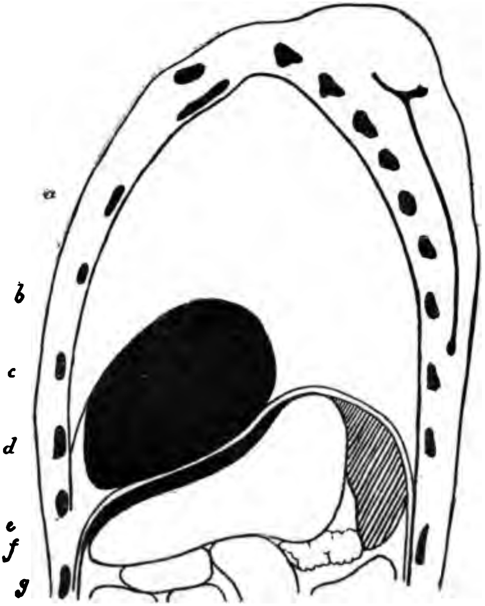

Fig. 22.—Antero-posterior section of thorax near the left parasternal line. (*See* text.)

(*Slightly modified from Luschka.*

It is also important to determine how much of the heart is quite uncovered by lung. This is done by continuing to percuss in the same direction as for the determination of the extreme limits of the organ, but with a lighter stroke, and observing when the slight resonance of the lung is replaced by absolute dulness.

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* The vagueness of the results obtained in this region is still further increased by the "sounding-board" property of the *sternum*.

The region of the heart covered by lung is called the **area of deep or relative cardiac dulness.** Its outline corresponds approximately to the anatomical outline of the organ. The region of the heart which is uncovered by lung and lies directly against the chest wall is called the **area of superficial or absolute cardiac dulness.**

The accompanying diagram (Fig. 22), which represents an antero-posterior section of the chest in the left parasternal line, will explain the sequence of phenomena which are observed on percussion. At *a* the resonance is full, at *b* it begins to grow emptier, and continues to do so till *d* is reached, when the sound becomes absolutely dull, and continues so over the uncovered surfaces of the heart and liver until *e* is reached, when the resonance of the stomach, though muffled, may be distinctly detected, and at *f* the muffled resonance gives place to the full tympanitic stomach note. The percussion stroke must be firm in passing from *a* to *b*, light from *c* to *d*, and light also as one percusses upwards from *g* to *e*.

1. **Deep dulness.**—By firm percussion, then, the right, the left, and that part of the upper border of the heart which lies to the left of the roots of the great vessels can be defined. To do this, percussion is performed in two directions: firstly, down a vertical line far enough from the middle plane to be quite to the left of the great vessels, but not so far out as to miss the upper border of the heart altogether—the left parasternal line, or one a shade internal to it, fulfils these requirements; secondly, percuss from right to left along a line as far down the chest as

possible, but yet clear of any trace of hepatic dulness; and also along the continuation of this line to the left of the heart, but this time percussing from left to right. In the vertical line one should begin at the first interspace, comparing its resonance with that of the second, and this with the third, and so continuing downwards until the first trace of impaired resonance is observed. One then knows that the boundary of the heart has been reached. But the dulness may also be present at the level of the rib above the interspace in which it was detected, and therefore the percussion sound of this rib must be compared with that of the one next above it. If the resonance of the lower of the two be less full than that of the upper, one knows that the outline of the heart lies behind it. The reason why rib is compared with rib, and interspace with interspace, is that otherwise the alternate presence and absence of the osteal resonance, according as rib or interspace is being percussed, introduces an element of unnecessary confusion. Before percussing the right border of the heart, the upper limit of deep hepatic dulness between the right parasternal and the right mammary lines must be determined, just as the upper border of the heart was. When this has been done, the right border must be defined by percussing from the right mammary line towards the sternum along the rib or interspace above the level at which the first traces of hepatic dulness were observed.

Although the lower border of the heart cannot be percussed out, a sufficiently close approximation to it is attained by drawing a line from the upper limit of deep hepatic dulness, which has already been determined, and which is usually found at the level of the fourth interspace or fifth rib, to the apex, whose position has been fixed by palpation.

By percussing in the fourth interspace from the left lung towards the heart, one is able to define the left border with considerable precision, and in cases where it is desirable to obtain further points one can percuss in various other lines perpendicular to the border, from the lungs towards the heart. The student must not confuse the slight muffling of the lung resonance which occurs when he arrives over the pectoral muscles with the deep dulness of the heart. It will be found that in an average healthy chest the percussion limits of the heart are as follow (Fig. 23):—

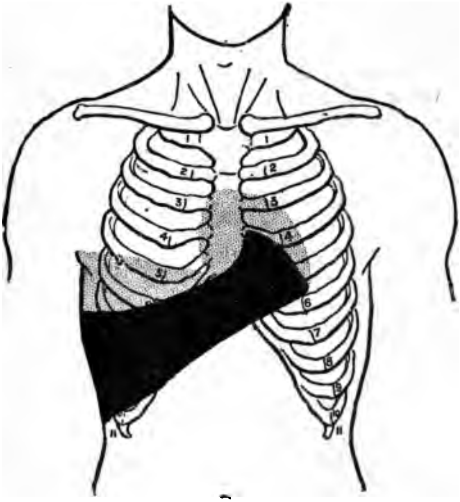

Fig. 23.—Superficial and deep dulness of normal heart and liver.

Upper border (in left parasternal line), at the third rib, or upper border of the third interspace.

Right border (at level of fourth rib) is just *to the right* of the right lateral sternal line. If, however, the lungs are somewhat voluminous, it may be impossible to detect the impairment of resonance at this point, and behind the sternum accurate observations are not very easy to make, as the sternum acts as a sounding-board, and collects vibrations from all the structures behind it, whether they lie directly beneath the point of percussion or are at some distance from it.

Left border (at level of fourth interspace), a shade internal to the mammary line. If it is percussed at a higher level it will be found to curve round so as to merge insensibly with the upper border.

Dr. Graham Steell has proposed the following convenient method of recording the position of the various boundaries of the heart: A vertical line is drawn, with a horizontal one lying across its upper end like the letter T. Above the horizontal line is recorded in Roman numerals the number of the rib to which the upper border of the heart extends in parasternal line. Below it, and to the writer's left of the vertical line, the distance to which the right border of the heart passes to the right of the mesial plane is noted in Arabic numerals which represent inches, whilst to the right of it the position of the left border of the heart is similarly indicated. The right border of the heart is determined along a line just clear of the liver dulness, the left immediately above the situation of the apex beat. Thus the record of an average healthy adult heart would be

$$\frac{\text{III.}}{1 \mid 3\tfrac{1}{2}.}$$

2. The **superficial dulness of the heart,** which depends on the position of the borders of the lungs, must be determined by light percussion. To ascertain the upper border, one should percuss down-

wards between the left lateral sternal and left parasternal lines. The left border is found by percussing from the left mammary towards the middle line along the fourth intercostal space, or fifth rib; the right, by light percussion at the same level, beginning to the right of the sternum. In health it will be found that the upper limit is at the level of the fourth rib. The left at its upper end is rather more than half an inch within the left border of the heart, as already determined; at its lower end it is decidedly nearer that border, and may extend outwards almost to the apex beat. The right limit does not correspond with the edge of the right lung, which, placed as it is behind the sternum, cannot be exactly defined, but lies in the left lateral sternal line, where it extends from the fourth to the sixth costal cartilage. The left limit curves gradually round to be continued into the upper, the latter joins the right limit at an angle. The space is therefore of triangular outline, but the left side of the triangle is not straight, but convex outwards. The lower side of the triangle cannot be defined by percussion, but corresponds with the inferior border of the heart; it is marked out in the manner already described.

Lying, as they largely do, behind the sternum, the dulness due to the great vessels can seldom be made out with precision. If, however, there is aneurysmal dilatation of the ascending aorta, a dull area can be mapped out. It is continuous below with that of the heart, above it bulges outwards to the right of the sternum at the level of the second interspace and adjacent ribs; whilst the sound produced by percussion of the manubrium sterni is also rendered much less resonant, or even, in cases where the aneurysm is large, absolutely dull *(Fig. 24).*

ALTERATIONS IN CARDIAC AREAS IN DISEASE.

In diseased conditions both the relative and the absolute cardiac dulness may be altered in size or in position. Except in cases of pericardial effusion the determination of the area of superficial dulness seldom yields any information of great importance regarding the heart; but it is valuable in revealing certain diseased conditions of the lungs and pleura.

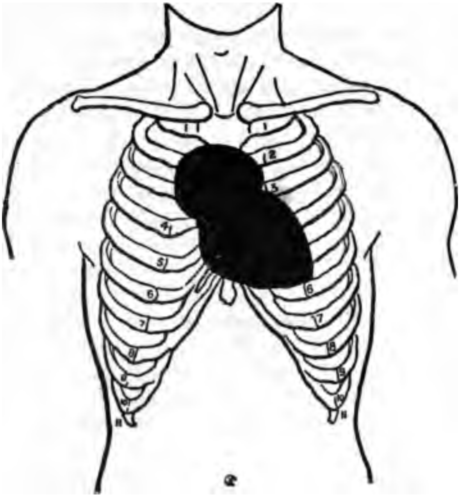

Fig. 24.—Aortic aneurysm.

When the relative or deep dulness is enlarged, the condition may be due either to disease of the heart and pericardium, or to morbid conditions in the surrounding viscera. If the dulness in the left parasternal line be found to extend **upwards** into the second interspace or higher, without any *corresponding* upward displacement of the lower

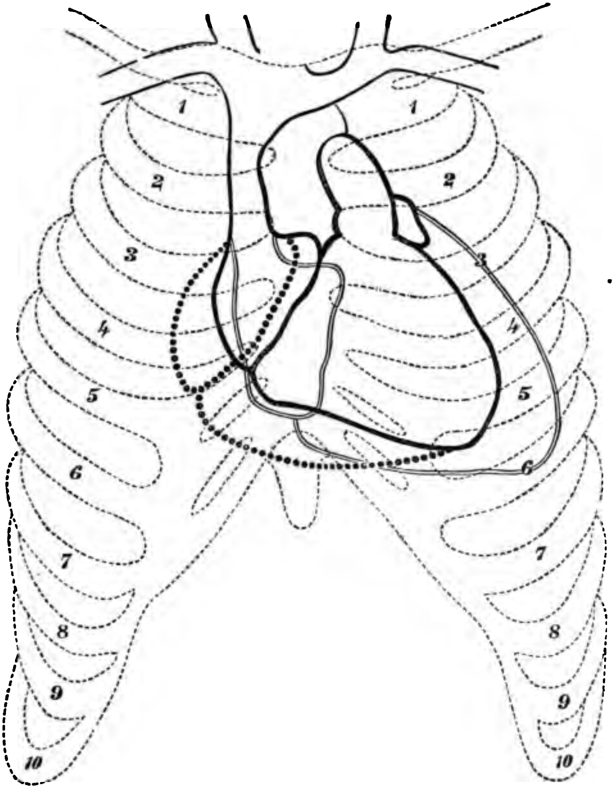

Fig. 25.—Diagram to illustrate the effect of dilatation of the right and left sides of the heart respectively. (*Gee after v. Dusch.*)

Continuous heavy outline, normal heart; dotted line, dilatation of right side; thin double outline, dilatation of left side.

boundary, such as would be present were the heart dislocated upwards as a whole, and in the absence of *disease of the lung*, the condition is usually due to *pericardial effusion.* Aneurysm of the descending arch of the aorta has been known to cause dulness in this region, but this effect is only produced in the rare instances where the aneurysm passes far forwards. Most aortic aneurysms are found farther to the right.

If the relative dulness extends to the **left of the apex beat,** provided the lung and pleura be healthy, we have to do with pericardial effusions; and in this case the right border will be found at a considerable distance to the right of the sternum—it may be as far as the right parasternal line. If the cardiac dulness extends to the left of the mammary line, but does not reach beyond the situation of the apex beat, the condition is probably due to dilatation and hypertrophy of the left ventricle, unless the *heart is bodily* dislocated to the left by some such

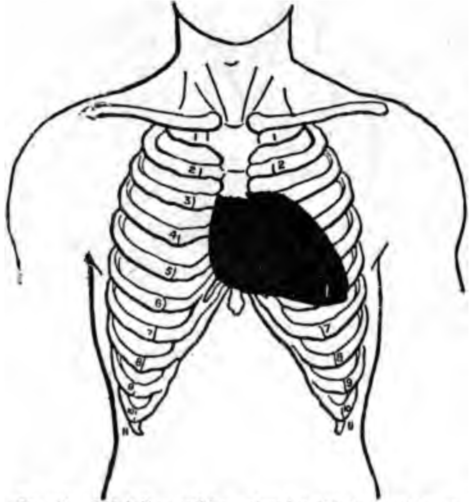

Fig. 26.—Dilatation and hypertrophy of left side of heart.

cause as massive pleural effusion on the right side. If the dulness extends, in the absence of lung disease, pleurisy, or pericardial effusion, more than about a finger's breadth to **the right of the sternum,** one is justified in concluding that the right heart has become dilated (Fig. 25).*

Since dilatation and hypertrophy of the left ventricle not only displace the left border outwards but cause depression of the apex, the percussion outline of such a heart will become conical (Fig. 26), whilst dilatation of the right side, by causing the cardiac dulness to extend too far to the right without greatly affecting the level of the apex, renders the dull area more square than usual (Fig. 27).

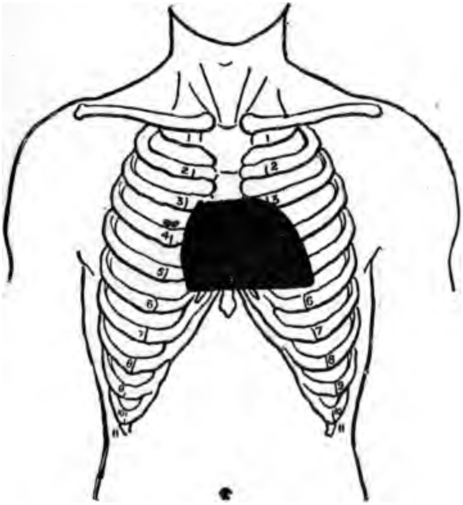

Fig. 27.—Dilatation of right side of heart.

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* The *auricle whose* boundary is thus determined is dilated, *the ventricle is usually* both dilated and hypertrophied.

The dulness which one finds in **pericarditis with effusion,** or in **hydropericardium,** varies with the amount of fluid which is present, but in well-marked cases is pear-shaped, with the broader end downward and the upper end higher than the ordinary upper limit of dulness (Fig. 28).

The chief causes outside the heart and pericardial sac that produce an increase in the area of cardiac dulness are due to diseases of the lungs and pleura. Here one may find consolidation or tumour of the lung, or pleural effusion; or a cirrhotic contraction of the lung, or the binding of it back by pleuritic adhesions, may leave more than usual of the front of the heart exposed, or at least near the surface. In comparatively rare instances the heart is pushed forwards *by a tumour* or aneurysm in the posterior mediastinum.

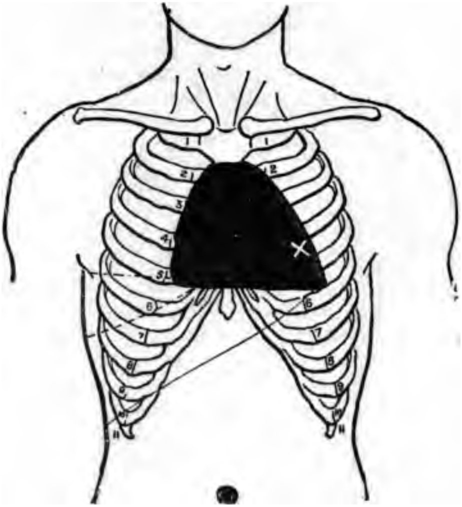

Fig. 28.—Pericardial effusion.

The relative dulness of the heart may be diminished in cases where the heart is unusually small, or the lungs so emphysematous as to interpose a layer of pulmonary tissue that is thick enough to carry the margins of the heart beyond the sphere of action of the percussion stroke. As a corollary to this, it should be observed that if, when the lungs are emphysematous, the heart's dulness reaches fully up to the normal limits, one is justified in assuming that, as a matter of fact, it oversteps these limits. Air in the pleural cavity will also diminish the area of dulness, whilst in pneumopericardium it is often completely abolished.

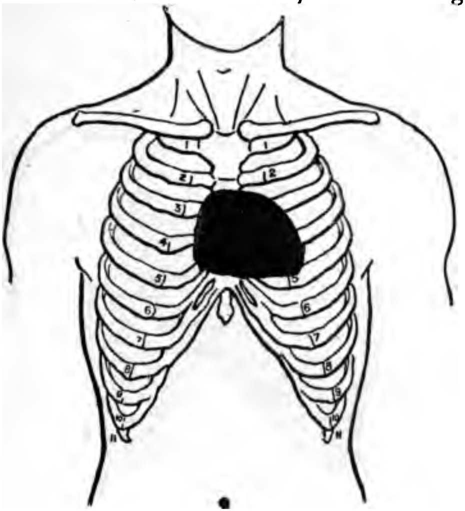

Fig. 29.—Displacement of heart upwards.

It is well to preserve a note of the breadth, in inches, of the cardiac dulness at the level of the fourth rib *or interspace.*

The area of absolute or superficial dulness is of less importance, being much affected by the state of the lungs. It is thus increased when they are retracted, decreased or almost abolished when they are distended, as in advanced emphysema; otherwise, it is altered by the same conditions and in somewhat the same manner as the area of relative dulness.

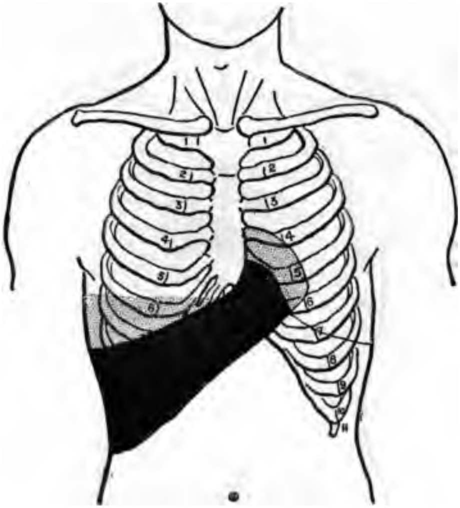

Fig. 80.—Displacement of heart and liver in emphysema.

The situation of the area of cardiac dulness is naturally altered by **changes in the position of the heart.** These result from its displacement by the pressure or traction of other organs, or from developmental anomalies. Thus in dexiocardia the heart is placed with its apex to the right, and the area of dulness is then the mirror image of what is usually found.

In cases of ascites or of massive abdominal tumour the heart is pushed upwards under the lungs. Hence its area of dulness is placed higher than usual, and, owing to the thickness of overlying lung, may be unusually difficult to map out (Fig. 29). Tumours of the liver displace the heart upwards and to the left. Pulmonary emphysema thrusts the heart

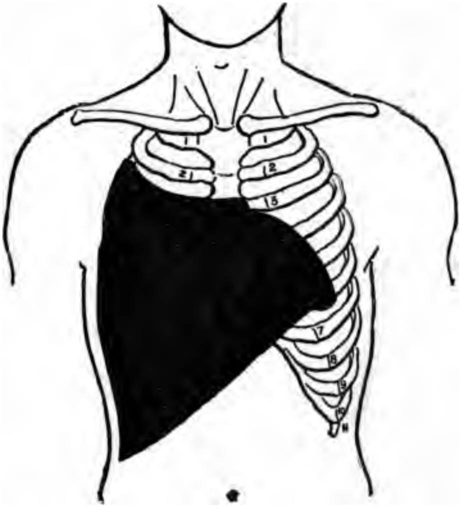

Fig. 31.—Displacement of heart in right-sided pleural effusion.

downwards (Fig. 30), pleural effusion drives it towards the sound side of the chest (Figs. 31, 32), whilst cicatricial contraction of the left lung often draws it upwards and to the left.

In certain cases of pericardial effusion it is stated that a small dull area can be found posteriorly near the angle of the left scapula. The phenomenon is of doubtful value, and has not been very satisfactorily accounted for. Those who wish to study it must refer to larger works or to special papers on the subject.*

Whilst the student is percussing the heart, he should attend not only to the resonance which is elicited, but also to the **sense of resistance** of the underlying tissues which the pleximeter finger experiences. By this means he may often form a shrewd guess of the nature of the subjacent structures. For example, a well-trained finger will detect decidedly greater resistance over a dull area when the dulness is caused by fluid, as in pleurisy or hydropericardium, than when it is produced by a solid organ like the heart or a pneumonic lung.

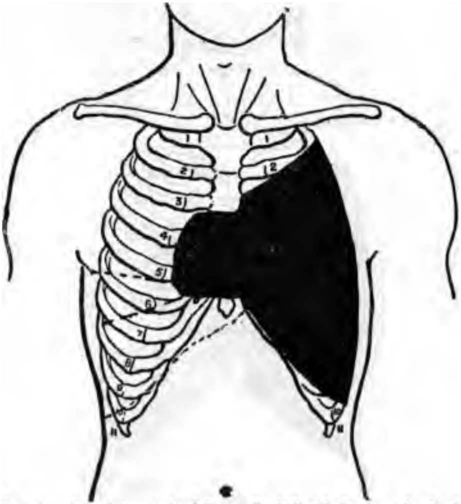

Fig. 82.—Displacement of heart in left-sided pleural effusion.

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* Sansom, "Diagnosis of Diseases of the Heart," p. 159; Ewart, *Brit. Med. Journal*, March 21, 1896, p. 717.

SECTION V.—AUSCULTATION OF THE HEART AND VESSELS.

Auscultation determines:—

(A) Character of the heart sounds with respect to
  1. Intensity.
  2. Rhythm.
  3. Quality.

(B) Abnormal sounds associated with the heart sounds
  (*a*) Over præcordia { endocardial murmurs. / pericardial friction.
  (*b*) Over vessels { clear sounds. / murmurs or bruits.

1. **The stethoscope.**—Auscultation, though sometimes performed by the direct application of the ear to the chest wall, is generally conducted by means of a stethoscope, and the student cannot take too great pains to choose a good one.

Stethoscopes are of two types, single and binaural. Each of these has its special advantages and disadvantages. Binaural instruments are particularly serviceable in the examination of children and of patients too ill to be much disturbed. Single ones, although they conduct less loudly, are relatively rather more sensitive to high-pitched sounds, and they also convey to the ear of the observer when he listens over the apex of the heart a distinct shock or jog at the moment of ventricular systole, thereby facilitating the timing of other phenomena. In the choice of a binaural, one should avoid instruments with unnecessary joints and loose parts, or with woven tubes. The chest piece should not be very large, nor made of metal; vulcanite is not so chilly, and is quite easily cleaned. It is obvious that unless the ear fittings are suitably shaped, much discomfort will be produced.

In choosing a single stethoscope much depends on the grain of *the* wood; some woods, such as oak, will be found to conduct better than others. The chest piece should be of moderate size, so that it may be in complete apposition with the chest wall even when the patient's ribs are prominent. The ear piece should be selected so as to fit the ear with comfort. The instrument should have no loose parts. A good rough and ready test of the efficiency of a stethoscope is to listen with it to the ticking of a watch, and to select the instrument which conducts the sound best. In practice the student must be very careful not to press heavily on the patient when using a single stethoscope.

The phonendoscope has not yet secured any very general recognition. This may be due in part to the fact that though it conveys low-pitched sounds with great clearness, it does not conduct high tones so well as the ordinary stethoscope ; and, since high tones are peculiarly characteristic of many morbid conditions in the lungs, it is obvious that its use for clinical purposes is seriously limited by this defect.*

2. The cardiac cycle and surface anatomy of the valves and vessels.

In order to understand the various sounds which can be heard by listening to the heart through the chest wall, a clear conception of the events which occur during a cardiac cycle is essential.

After the completion of a beat the auricles and ventricles are both relaxed. Thereafter the auricles contract, forcing their contents through the cuspid valves into the ventricles, and filling them. The ventricles then contract in turn, expelling the blood into the vessels, whilst the auricles commence to relax and become refilled with blood; finally, the ventricles relax also, and so the cycle is completed. We have thus in rapid succession auricular systole, ventricular systole, and ventricular diastole; the auricular diastole commencing during ventricular systole, and ventricular diastole continuing through auricular systole.

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* A special stethoscope provided with an adjustable slit has been devised for the purpose of comparing the intensity of the different heart sounds at varying points. By adjusting the opening until the sound under examination becomes inaudible, one can obtain a definite measure of its intensity at any given point. The best form of this instrument is Bock-Oertel's stethophonometer, which is a modification of the pattern *originally suggested* by Gärtner.

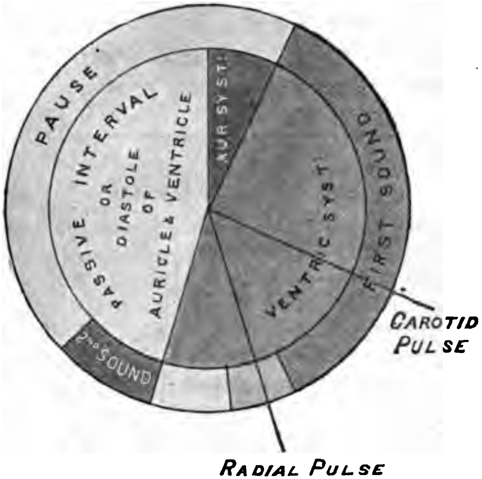

Fig 83.– Cardiac cycle. (*Modified from Gairdner.*)

The beginning of ventricular systole is marked by the closure of the mitral and tricuspid valves, which had remained open during the systole of the auricles, and by the occurrence of the apex beat; the beginning of ventricular diastole is marked by the closure of the aortic and pulmonary valves, which remain closed until the beginning of the following ventricular systole. The pulse in the carotid occurs a short time after the *commencement* of ventricular systole; in the radial artery it is decidedly later in its appearance, and therefore the radial pulse must never be taken as an index to the commencement of ventricular systole. The carotid pulse is less fallacious than the radial, but, as an index to the commencement of systole, preference should be given to the apex beat whenever it is available.

Various authors have constructed diagrams to represent the sequence of events in a cardiac cycle. The accompanying one (Fig. 33) may be taken as representing these in an ordinary case, though the relative duration of the successive events will be found in practice to vary within fairly wide limits. The most important variation is that when the heart acts with unusual rapidity the duration of diastole is curtailed to a greater degree than that of systole, and hence a shorter interval elapses between the time of closure of the semilunar valves and the commencement of ventricular systole than one would infer from an examination of the diagram. For some purposes it is found more convenient to unroll the above diagram, so that the sequence is represented along a straight line instead of round a circle. When this is done the scheme takes the form represented in Fig. 34.*

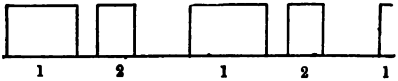

Fig. 34.

1, First sound ; 2, second sound.

In addition to a knowledge of the cardiac cycle, auscultation presupposes acquaintaince with the situation of the valves of the heart and of the course of the principal arteries, as well as of the areas where sounds produced at the valves are best heard. For full particulars the student must consult works on

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* It must be recollected that in clinical language the words systolic and diastolic are used with reference to the state of the ventricles, events which take place during the auricular contraction being described as diastolic (or pre-systolic). On the other hand, physiologists generally regard the period of auricular contraction as included in the systolic period of the cardiac cycle.

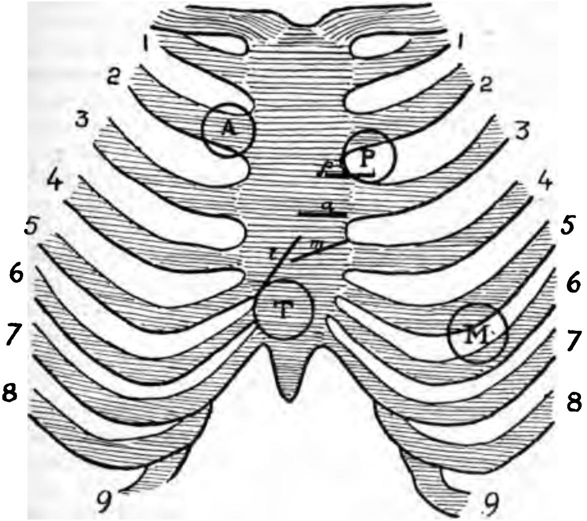

Fig. 35.—Position of the cardiac valves and auscultatory areas.

regional anatomy. The following summary merely recapitulates the most important facts :—

The **pulmonary valve** lies horizontally at the level of the upper border of the third left costal cartilage; the right half of the valve lies under cover of the sternum, the remainder passes outwards behind the costal cartilage (Fig. 35).

Clinically, then, the systolic phase of the cycle begins with the apex beat and commencement of the first sound. It terminates immediately *before* the second sound, whose commencement *marks the beginning* of the diastolic period.

The **aortic valve** lies farther from the surface, and at a slightly lower level. Its situation may be indicated on the front of the chest by a line drawn horizontally across the left half of the sternum on the level of the lower border of the third costal cartilage.

The **mitral valve** lies slightly obliquely behind the inner end of the fourth left costal cartilage and adjoining part of the sternum. The **tricuspid valve** is placed much more obliquely; its upper end is opposite the fourth cartilage or interspace, and its lower near the lower border of the fifth right costo-sternal articulation. It marks the line of junction between the right auricle and right ventricle.

The **pulmonary artery** is situated at the inner edge of the second left interspace, and behind the adjacent part of the sternum. At the lower border of the second cartilage it divides into its branches to the right and left lungs.

The **ductus arteriosus** passes upwards from the left branch to join the aorta.

The **aorta** arises behind and slightly lower down than the pulmonary artery, and, passing upwards and to the right, approaches the surface of the chest most closely at the inner end of the second right costal cartilage, arching backwards and to the left from that point. The **innominate artery** passes in a direction represented by a line drawn from the middle of the manubrium to the right sterno-clavicular junction.

A stethoscope placed over the valves of the heart would fail to distinguish at which of them a given sound takes origin, because they lie so near each other that the sounds from all of them would reach its chest piece. Besides, in the case of the valves that lie more deeply the sounds would have to pass through the chambers of the heart which are situated between them and the surface, and thereby their clearness would be impaired. To avoid these inconveniences, the sounds produced by each valve are listened for over that part of the chest where the cavity in which the valve lies most closely approaches the surface, and is most remote from the other cavities of the heart.

Hence one listens to the mitral valve at the cardiac apex, to the tricuspid at the lower end of the sternum, to the aortic over the aorta at the second right costal cartilage, and to the pulmonary over the artery in the second left intercostal space. It is practically found that in these regions the sounds of the respective valves are heard with a maximum of loudness and distinctness. They are therefore called **the mitral, tricuspid, aortic, and pulmonary areas,** although they do not lie immediately over the valves from which they derive their names. Auscultation should be performed systematically over these areas. In ordinary cases the student may begin with the mitral area, making certain of the time at which the sounds that he hears occur in the cardiac cycle by feeling the apex beat whilst he listens. He may then pass to the tricuspid area, thereafter to the aortic, and lastly to the pulmonary. When necessary, auscultation may also be performed along a diagonal line joining the mitral and aortic areas. This is often of service, as, for instance, when a mitral systolic murmur is associated with an aortic one.

In health two sounds are often heard over each of these areas, the first corresponding with the beginning of ventricular systole, the second with the commencement of ventricular diastole. The first sound depends, from the clinical standpoint, chiefly on the closure of the mitral and tricuspid valves, and to a lesser degree on the muscle tone of ventricular contraction, on which, however, its duration depends, and on other subsidiary causes. The second sound is due to the closure of the aortic and pulmonary valves, and also, but very subordinately, to tension of the vessel walls. This sound is sharper and shorter than the first, which continues through an appreciable period of systole, but not until its termination. At and to the left of the apex, only the aortic element of the second sound is audible. The observer must remember that it is always important to note the character of both the first and second sounds in each of the areas. In disease the following deviations from the normal may occur :—

1. The sounds may have a different intensity, both absolutely and relatively to each other, from that which they possess in health. In estimating this, allowance must be made for the thickness of the chest wall and the volume of the lungs.

2. The sounds may be doubled, or their rhythm altered.

3. Adventitious sounds may be heard, either replacing or occurring along with the heart sounds.

1. ALTERATIONS IN INTENSITY.

(*a*) **The first sound may be weaker than usual.** Decided shortening or weakness of the first sound, still more its disappearance, indicates cardiac failure. In acute febrile disease this change may occur rapidly, and should always be looked for; the left side of the heart generally yields first.

(*b*) **The first sound may be louder than usual.** It is then said to be **accentuated.** In *simple dilatation* the sound is often slightly accentuated and very clear, sharp, and short, as a large volume of blood is projected against the valve curtains, and the sound easily penetrates the thin ventricular wall to the observer's ear. In *hypertrophy* the sound is accentuated, but dull, prolonged, and *thudding,* as the vibrations produced by the unusually forcible closure of the valve have to pass through the thickened heart walls, and the muscular element of the sound is specially prominent.

(c) **If the second sound is more distinct** in the mitral or tricuspid areas than the first, we have either to do with a weakened first sound or an accentuated second; whilst if the first sound is louder than the second in the aortic and pulmonary areas, the first sound is accentuated.

(*d*) **The relative loudness** of the second sound in the aortic and pulmonary areas varies somewhat, and is a good deal influenced by the patient's age. The pulmonary sound is rather more accentuated than the aortic in youth; in old age the reverse is the case* when the subject is in good health.

Accentuation of the second sound means that the valve where the accentuated sound is produced is closed with unusual force. The force of closure depends on the momentum of the column of blood that effects it, and the momentum depends equally on the mass of moving blood and on the velocity of its recoil against the valve. In the aorta the mass of blood is increased when the vessel is dilated near its origin; the velocity of recoil when, in consequence of contracted arterioles or other obstruction to the outflow of blood, the arterial blood pressure is increased. When the aortic accentuation is due to the former cause, the sound often assumes a peculiar resonance suggestive of the echo produced when a cork is drawn from an empty bottle. Over the pulmonary artery an accentuation of the second sound generally indicates increased blood-pressure in the pulmonary circulation, due to disease either of the lungs or of the left side of the heart. *In pneumopericardium* the sounds are singularly clear and resonant, *in pericardial effusion* they are faint and muffled, *in pneumothorax* they are in many instances accentuated and clear, whilst when there are *cavities in the lung* near one of the valve areas the sound arising in that area may be reinforced by the cavity acting as a resonator.

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* *Cf.* Beneke: "Die Altersdisposition," plate 1; also, Creighton, "On the Relative Intensity of the Second Sound of the Heart at its Base" (1,000 cases), New York *Medical Record*, Jan. 13th, 1900; and Ewart, "On the Normal Auscultation of the Heart," *Lancet, 1893, vol.* i., p. 1241.

2. REDUPLICATIONS.

Under certain conditions the first or the second sounds may be **doubled.** Various explanations have been offered to account for the reduplication. The simplest is that which assumes that when such a doubling occurs, the valves, either cuspid or semilunar as the case may be, close sooner on one side of the heart than on the other; the reason being, in the case of the mitral and tricuspid, asynchronism of systole of the respective ventricles, and in the case of the aortic and pulmonary valves some variation of the normal relations of blood pressure in the aorta and pulmonary arteries, since an increase in pressure will accelerate the closure of the valve which is subjected to it. This view is probably correct in some, but not in all cases. Clinically, reduplication of the first sound occurs under very various conditions, the most important variety being known as the "**bruit de galop,**" where the rhythm at the apex shows the accent on the second element of the triplet ◡ –́ ◡. In some cases the appearance of this "bruit de galop" is of very unfavourable import.* Occasionally it is difficult to discriminate between a short presystolic murmur and a reduplication of the first sound. This difficulty is most apt to occur when the heart is acting feebly, and it is often found to clear up after a few days of rest and treatment. An experienced observer will seldom be at a loss, however, in determining to which class such a case should be referred.

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* *Cf.* Fraentzel, "Vorlesungen über die Krankheiten des *Herzens,*" *part i., p. 56.*

Reduplication of the second sound indicates, in a large proportion of the instances in which it is heard, an increase of pressure in the pulmonary circulation. It occurs, therefore, in certain lung diseases and in diseases of the left side of the heart, being very characteristic of mitral stenosis, in which disease it is heard in a large proportion of the cases. It should, however, be mentioned that many observers do not consider that the reduplication in mitral stenosis is due to early closure of the pulmonary valves.* It is also found where the right and left ventricles fail to contract simultaneously, whether the failure be due to an increase of work thrown upon one of them, or to the presence of structural changes in the heart muscle, or to derangement of the nervous mechanism which regulates their action. It also occurs physiologically at the end of a full inspiration and the beginning of the following expiration, being in this respect the converse of the first sound, which is more often reduplicated at the end of expiration and the commencement of inspiration. When reduplication of the second sound is observed, it is most important to determine its presence or absence, and its character in each of the four valve areas.

ALTERATIONS IN RHYTHM.

Alterations in the rhythm of the sounds deserve attention.† The usual rhythm is that of triple time in music, with the accent on the first beat in the mitral and tricuspid areas, and on the second in the aortic and pulmonary, whilst the third beat is silent. This is slightly modified by the fact that a quickly-acting heart gains time chiefly in the period of ventricular diastole, but the relation of the sounds is less modified by this than the phases of the cycle are. When, however, the vitality of the heart has been seriously impaired by long-continued high blood tension, such as is seen in chronic nephritis, and especially if fever or some such cause assists in weakening the myocardium, the sounds become amost equidistant, the period of ventricular systole being unduly lengthened.

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* *Vide infra*, p. 161.

† For fuller information, consult Broadbent's "The Pulse," p. 63.

This *deliberate pendulum-like sequence* of the sounds should always be regarded with considerable anxiety, as it points—unless, indeed, the patient is being overdosed with digitalis—to serious involvement of the cardiac muscle.

Exactly the opposite effect is produced on the rhythm when systole is rapidly accomplished, or when, from great weakness, the ventricle fails to effect a complete emptying of itself, and the systole becomes abortive.

3. ADVENTITIOUS SOUNDS.

Adventitious sounds may be of three kinds: endocardial, vascular, or exocardial. Abnormal endocardial sounds are called **murmurs,** or bruits. They are due to disease either of, or close to, the valve where they occur, when they are often known as organic; or to some alteration in the state of the blood, which, by affecting its viscosity on the one hand and the nutrition of the tissues of the heart and vessels on the other, produces the conditions necessary for the development of a murmur.

The physical explanation of murmurs is by no means simple. The following are some of the factors concerned in their production:—

1. The viscosity of the blood;
2. The velocity of the blood-stream;
3. The passage of the stream from a narrower into a wider channel.

The third condition is equally well produced when a narrowed orifice leads to a normal cavity beyond it, or when a normal orifice opens into a dilated cavity.

Endocardial murmurs always have a definite relation to the events occurring in the course of the cardiac cycle, their time and import varying with their point of origin.

In the so-called "organic" cases, where the valves or their surroundings are implicated, a murmur may either result from obstruction to the onward flow of the blood, or from leakage backwards through a closed but incompetent valve. The former are known as obstructive murmurs, the latter as regurgitant. In examining a murmur the following points must be noted:—

(*a*) Its time of occurrence;
(*b*) Its point of maximum intensity;
(*c*) Its direction of selective propagation beyond the præcordial area;
(*d*) Its character.

The **time of its occurrence** is noted with reference to the sounds of the heart, and these by comparison with the time of occurrence of the apex beat.

The **maximum loudness of a murmur** which has been produced at a given valve usually occurs at the point where the valve sound would be best heard in health. To this rule, however, there are some exceptions.

Experience shows that valvular murmurs are not equally well heard at all points of the chest wall which are equidistant from the point of their greatest intensity, but that each is much more distinctly audible at a distance in some directions than in others; this fact is expressed by saying that such murmurs have **directions of selective propagation.***

The **character** of the murmur also helps to decide a doubtful case. Obstructive murmurs are apt to be rough; regurgitant to be soft and blowing.

The pitch and general quality of murmurs vary greatly; some have quite a distinct musical character, others are harsh and sawing. The loudness of a murmur has no relation to the amount of damage which causes it. A very loud murmur is often far less serious than one so soft as to be nearly inaudible.

Murmurs due to disease of extra-uterine origin are very much oftener found to proceed from the valves of the left side of the heart than from those of the right, and in adult life murmurs at the tricuspid and pulmonary areas, due to morbid processes arising in these valves, are rare. The following is a short epitome of the chief murmurs which may be heard at the various valves: the diagrams illustrate the position of the more common ones in the cardiac cycle.

(1.) MITRAL MURMURS.

Mitral murmurs may be either obstructive or regurgitant (Fig. 36).

(*a*) **Obstructive** murmurs occur during ventricular diastole, and are invariably of organic origin. They sometimes follow immediately on the second sound, when they are known simply as "*diastolic.*" At other times the murmur is separated from the second sound by a brief interval, but terminates before the occurrence of the first sound; it is then called "*mid-diastolic*"; in yet other instances the murmur only begins with the advent of auricular contraction, when it is designated an "*auriculo-systolic,*" or, more often, a "*presystolic*" murmur. In each case the murmur is due to the onward rush of the blood through the deformed or narrowed mitral valve into the wider cavity of the left ventricle. During the earlier part of diastole this is effected by the aspiration of the relaxing ventricle, which is ordinarily strongest near the beginning of diastole—at the end of the period the contracting auricle is the main agent in producing the flow. Sometimes an early diastolic murmur is followed by a moment of silence, which is then succeeded by a presystolic murmur.* Best heard at the apex, or sometimes rather nearer the sternum, they have no direction of selective propagation; they

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* The complete explanation of the selective propagation of many murmurs is still unknown. In most cases, however, the following factors greatly influence the direction in which the murmur is conducted:—(1) The varying conductivity of the different structures in the chest wall, and between the chest wall and the heart; (2) the direction of the vibrating bloodstream; (3) the position of the chamber of the heart or of the vessel in which the valve lies.

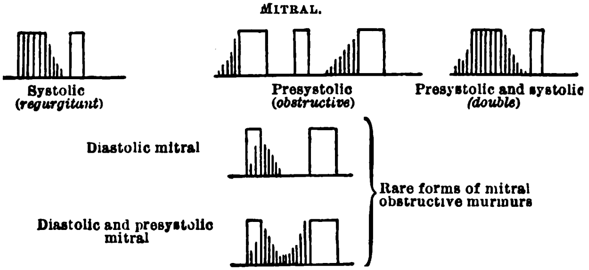
MITRAL.
Systolic (*regurgitant*)
Presystolic (*obstructive*)
Presystolic and systolic (*double*)
Diastolic mitral
Diastolic and presystolic mitral
Rare forms of mitral obstructive murmurs

Fig. 36.—Mitral murmurs. (*After Wyllie.*)

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* The term "post-diastolic" as applied to certain of these murmurs is very misleading. They are all diastolic in time, though, as has been stated, some are separated from the second sound by a pause.

are harsh and rough in character, more particularly when of the presystolic variety, and very often are associated with a distinct thrill. In the majority of cases the second sound is reduplicated, so that the murmur and accompanying sounds may be phonetically represented by "*rr*úp ti-ti"; or, where the heart's action is rapid and the murmur occupies a considerable portion of the diastolic period, by "ti-ti *rr*úp." Occasionally the presystolic murmur is accompanied by a mitral obstructive murmur occurring at the beginning of diastole, when the phonetic representation would become "*rr*úp ti tiff *rr*úp ti tiff," or, if the murmur occur a shade later in diastole, by "*rr*úp titi iff, *rr*úp titi iff."

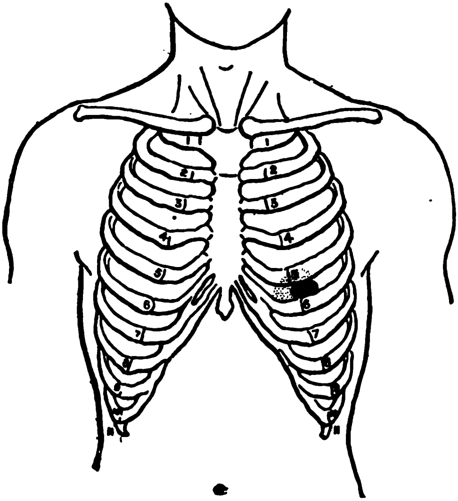


Fig. 87.—Presystolic mitral murmur

The exact significance of the reduplication of the second sound in mitral obstruction is a matter of dubiety. At present many observers deny that the second element of the reduplication is produced at the semilunar valves, and the fact that it is better heard at the apex than at the base supports their contention. It is not improbable that one element of

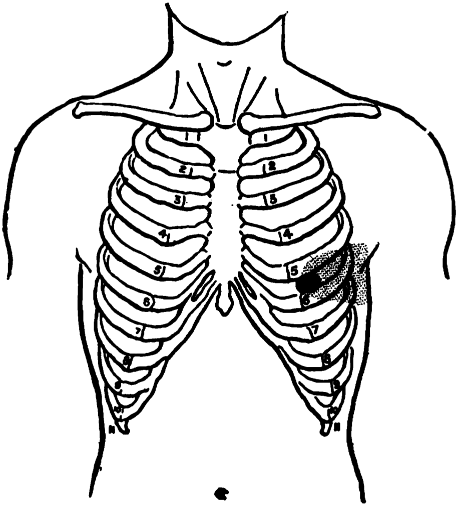


Fig. 38.—Mitral systolic murmur—propagation in front.

the double sound under discussion may originate in the mitral valve, whose segments, partially adherent along their adjacent margins, are no longer free to fall backwards before the blood-stream as it again passes from the auricle to the ventricle at the commencement of diastole, but are suddenly arrested in their progress, and, bulging into the ventricular cavity, become tense, and emit a sharp sound like that produced by a sail suddenly *bellied* by a gust of wind (Fig. 37).

(*b*) **Regurgitant murmurs** occur during ventricular systole, and may be either organic or simply due to dilatation. They begin with the apex beat and replace more or less completely the first sound in the mitral area. Their point of maximum intensity is at the apex, their direction of selective propagation is outward towards the axilla and angle of the left scapula, and they are generally soft and blowing in character. Slight mitral systolic murmurs, especially those due to dilatation of the ventricle and mitral orifice, and not to disease of the valve curtains, frequently lack any selective propagation backwards. It is possible that some basal hæmic murmurs may be due to mitral regurgitation when the left ventricle is *dilated (see p.* 168) (Figs. 38, 39).

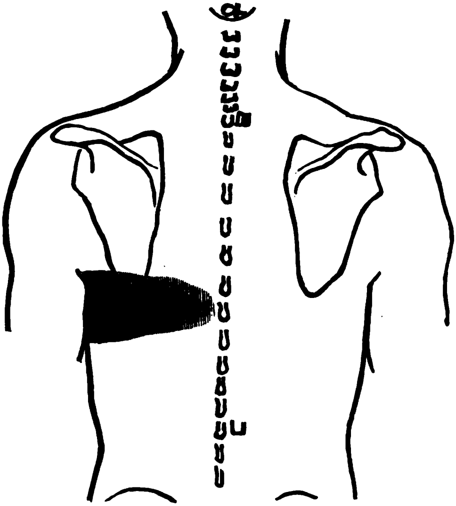
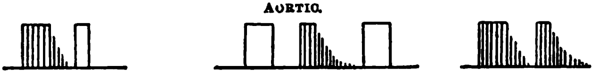


Fig. 39.—Mitral systolic murmur—propagation behind.

(2). AORTIC MURMURS.
(Fig. 40.)

(*a*) **Obstructive murmurs** occur during ventricular systole; they are due either to obstruction of the ostium aortæ from valve disease or to aortic dilatation beyond a normally-sized ostium. They are rough in character; have their area of greatest loudness at the second right costal cartilage near the sternum; are propagated with the blood-stream into the arteries; and may, in most instances, be readily heard over the carotids—sometimes at a much greater distance (Fig. 41).



Systolic (*obstructive*) Diastolic (*regurgitant*) Systolic and diastolic (*double*)

Fig. 40.—Aortic Murmurs. (*After Wyllie.*)

(*b*) **Regurgitant murmurs** occur during ventricular diastole; they begin with the closure of the semilunar valves, and replace in part or completely the normal second sound in the affected region. They may be best heard in the aortic area; not unfrequently, however, they are as distinctly audible over the left half of the sternum, at the level of the third rib and interspace. Their direction of selective propagation is towards the lower end of the sternum, though occasionally they are almost equally well heard near the apex; their character is less harsh than that of systolic aortic murmurs. Their intensity is greatest at first, and gradually diminishes during the diastolic period. Marked regurgitation leads to auscultatory phenomena in the arteries (*see* p. 170) (Fig. 42).

In many instances one finds that a **double murmur** is present at the aortic orifice, the systolic element of which is not caused by real stenosis of the ostium, but by roughening and deformation of the valve segments, the diastolic murmur being due to the backward leakage through the misshapen cusps. This double murmur often possesses a very distinctive "sawing" character.

Austin Flint has directed attention to the presence of a presystolic murmur at or near the apex of the heart in certain cases of aortic disease where at the *post-mortem* examination, no change was found to be present in the mitral valve. Most probably this murmur indicates a dilatation of the left ventricle so considerable as to render the mitral orifice relatively too narrow for the cavity beyond.

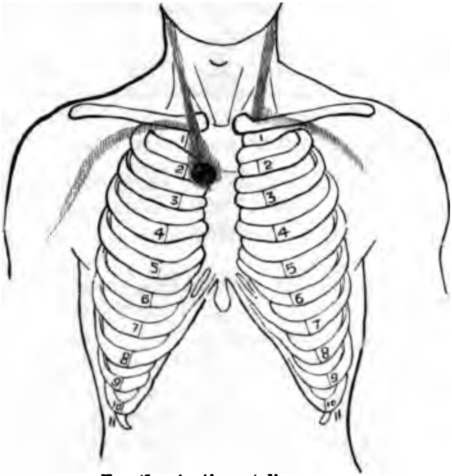


Fig. 41.—Aortic systolic murmur.

(3). TRICUSPID MURMURS.

These are comparatively rare.

(*a*) **Obstructive murmurs** resemble those of *the mitral valve,* but have their maximum intensity at the lower end of the sternum. They have no selective propagation.

(*b*) **Regurgitant murmurs** have a similar character to mitral regurgitant murmurs, are best heard in the tricuspid area, and are associated with the venous pulse, *q.v.* (p. 194). They are usually a sequel to disease of the left side of the heart, after compensation has failed.

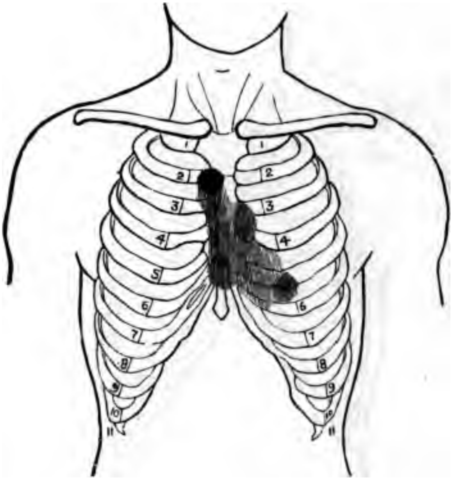


Fig. 42.—Aortic diastolic murmur.

(4). PULMONARY MURMURS.
(Fig. 43.)

These are best heard in the pulmonary area, have no direction of selective propagation, are usually systolic, though occasionally they are well heard as high as the first rib, and are rarely due in extra-*uterine life to* disease of the valve, but are most often caused by dilatation of the artery beyond the valve ring. They are very well heard in many cases of exophthalmic goitre.* A diastolic pulmonary murmur is excessively rare.

(5). MULTIPLE MURMURS.

In a large number of cases **more than one murmur** is present during the cardiac cycle. When they occur at different epochs, it is easy to study each separately; when two or more occur together, each will be found to possess its own position of maximum loudness, its characteristic direction of selective propagation, and its peculiar quality of sound (Fig. 44). Each lesion, moreover, will produce more or less definite

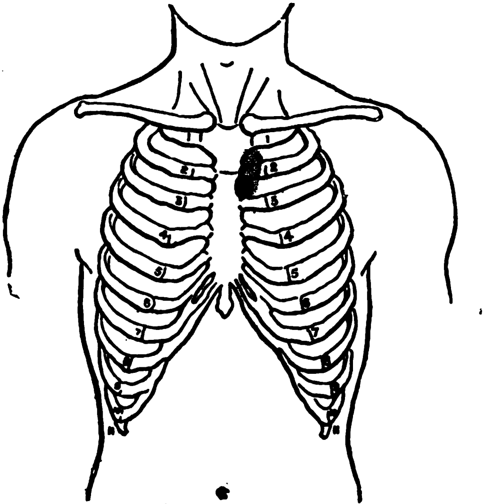


Fig. 43.—Pulmonary systolic murmur.

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* *In these cases there is often an aortic systolic murmur also.*

effects on the general circulation, and by observing these by the other methods at our disposal a diagnosis can usually be arrived at. It must be recollected that during the last few days of life, when the diseased heart is acting feebly, serious lesions are often unaccompanied by any murmur, the force of the blood-stream being too weak to produce any vibration. Some murmurs vary very much with the attitude of the patient; it is therefore important to examine the heart both in the erect and supine positions.

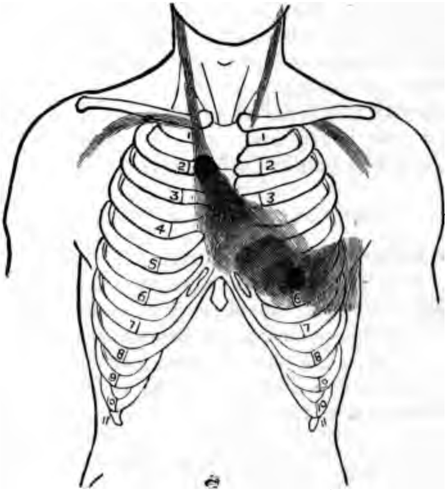

Fig. 44.—Combined aortic and mitral systolic murmurs.

(6). CONGENITAL MURMURS.

A **patent foramen ovale** may give rise to a murmur at the base of the heart, dependent on *difference of* pressure in the right and left auricle; a murmur usually rather harsh in character, systolic in time, and with its maximum intensity at the level of the second left costal cartilage and first interspace, a short distance outwards from the sternum, is sometimes caused by the **ductus arteriosus** remaining unclosed. The diagnosis of congenital cardiac murmurs is often fraught with considerable difficulty. The following points may be noted regarding them. They are mostly systolic, the situation of maximum intensity and the direction of propagation do not correspond with those of ordinary murmurs, whilst the fact that they are often associated with humming bruits at the base of the heart, and with an abnormal cardiac contour, may assist in their recognition.*

HÆMIC AND VASCULAR MURMURS.

In anæmia several murmurs are frequently heard over the heart and vessels. One, which is of specially common occurrence, is audible in the second left intercostal space over or just external to the pulmonary area. Various theories have been advanced to account for it. The chief of these are—

1. **Naunyn's theory,** where it is held that the sound is due to regurgitation through the mitral valve into the left auricle, and that it reaches the ear by way of the auricular appendix, which for various reasons—including the fact that the anterior border of the left lung is often rather retracted in anæmia—approaches the chest wall more closely than under other conditions. Naunyn and others have pointed out that the murmur attains its maximum intensity not over the pulmonary artery, as is found in undoubted cases of pulmonary systolic murmurs, but farther to the left; and the writer's observations confirm this statement in a preponderating majority of cases.

2. **Russell's theory.**—This theory assumes that the left auricle is dilated and filled with blood at unusually high

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* The student will find an admirable epitome of what is known regarding congenital defects in the heart in an article by Dr. John Thomson in the "Encyclopædia Medica" (s. v. Heart).

pressure, and that it therefore presses sufficiently firmly against the pulmonary artery to produce a constriction. The hypothesis is unsupported by any cogent facts, and is inconsistent with a sound view of the mechanics of the circulation.

3. Many writers regard the murmur **as due to slight dilatation of the pulmonary artery** beyond the valve ring, and to a less viscous condition of the blood, which would accentuate the vibrations set up by such a dilatation.

4. **Sansom*** holds that the vibrations which cause the murmur arise in the conus arteriosus, and perhaps also in the semilunar valves themselves, and are due to a fibrillary tremor of the over-strained muscular fibres which are found in these regions.

The first and third of the above theories have at present the greatest number of supporters.

Hæmic murmurs are also heard at times in the mitral, and much less frequently in the tricuspid and aortic areas, the last being particularly uncommon. *In all cases such murmurs are systolic in time.*

A continuous humming sound is often audible over the veins at the root of the neck in chlorosis. It is known as the **bruit de diable,** and is caused, in part at least, by an alteration in the calibre of the internal jugular vein as it passes through the cervical fascia.

In order to hear the *bruit de diable* clearly, the stethoscope must be held very lightly, so as to exert no pressure, over the clavicular head of the sterno-mastoid muscle. Not seldom one may also hear the sound perfectly distinctly when the stethoscope is placed on the sterno-clavicular articulation, by which manœuvre all possibility of creating a factitious bruit by pressure is avoided.

Hæmic murmurs may arise **in the larger arteries,** and are present independently of the pressure of the stethoscope, and it seems probable that an important, if not the chief, factor in their production is a disturbance of the vasomotor mechanism of the vessels.*

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* "*Diagnosis* of Diseases of the Heart," p. 285.

There are, moreover, other sounds which may become audible in the arteries, and which are the result of changes in the pressure of the blood-stream. The most notable instance of this is found where relaxed arteries are so rapidly distended by a large blood-wave that their walls are thrown into vibration by the sudden strain, and a sound is produced which corresponds with the advent of the pulse-wave. In cases of aortic regurgitation, where these conditions are most fully developed, we have also a second sound which occurs at the instant when the pressure once more falls off. This double sound, when heard in the femoral, is very characteristic of aortic regurgitation. Pressure produced by an ill-applied stethoscope often converts these sounds into murmurs.

A murmur in the subclavian artery as it crosses the apex of the lung may be caused by pulmonary disease.

When there is an **aneurysmal dilatation** of the aorta, murmurs may or may not be present, or the aortic second sound may be accentuated over the sac; but no definite rule holds for such cases. When an aneurysm opens into another large vessel—such, for example, as the superior vena cava—the murmurs produced may be very loud, and are heard in unusual situations.

EXOCARDIAL SOUNDS.

Exocardial sounds may be due either to pericardial friction or to a localised pleurisy near the heart.

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* A valuable contribution to our knowledge of the physical causes which lead to the production of hæmic bruits is to be found in Thoma's "Textbook of General Pathology" (English edition, vol. i., p. 271). Compare also Nicolls, *Jour. Physiol., vol. xx., p. 413.*

When **pericardial friction** occurs over an area uncovered by lung, it has a singularly superficial character, and thus can often enough be readily recognised.

Unlike the murmurs already described, pericardial friction does not correspond definitely with the events of the cardiac cycle. It is generally more distinct in systole than in diastole, but tends to exhibit a to-and-fro character, the first element occurring during systole and the second during diastole, but not necessarily commencing at the beginning of either phase. Sometimes the sound occupies the latter part of systole and the early part of diastole without exhibiting any pause between its first and second elements; sometimes it remains audible during the whole of the cardiac cycle. Further, its position of greatest intensity does not correspond with any of the areas in which valvular murmurs are best heard, and it is not propagated to a distance, but remains confined within narrow limits. Its position may be observed to vary from day to day. As a rule, it appears first near the base of the heart on the left side, but when the condition has become general it is best heard near the left nipple, and is sometimes associated with a distant thrill. The intensity is often considerably modified by the attitude of the patient. When the inflammatory process involves the auricle as well as the ventricle, the to-and-fro rub may be replaced by a triple friction sound.

Fig. 45.—Pericardial Friction. (*After Wyllie.*)

A. Ordinary type, one rub in systole and one in diastole.
B. Type with triple rhythm, one rub in systole and two in diastole.

In *pericarditis* the heart's action is apt to become tumultuous. *When fluid is poured out, the cardiac sounds become faint and distant.*

When air and fluid are both present in the pericardial sac—an event of very rare occurrence—a churning or "water-wheel" sound can be heard on auscultation.

To distinguish between the rub of pericarditis and that of pleurisy over a neighbouring portion of lung, the patient should be instructed to hold his breath. Pericardial friction is unchanged by this, but, if it be of pleural origin, it will either be much reduced in intensity or will wholly cease. On the other hand, deep respiration will increase the pleural sound, but will not influence the pericardial.

The possible co-existence of both pleuritic and pericardial friction must not be overlooked.

SECTION VI.—THE PULSE.

The examination of the pulse gives us direct information regarding two things, namely, the condition of the vessel walls and the amount and variations of pressure of the contained blood. By intelligent observation of these facts we can obtain very valuable information regarding the state of the heart and circulation, as well as the general state of the patient.

When any observation is to be made on the pulse, the patient should be lying on his back, or at least sitting; and, except for special purposes, should not have been making any effort for some little time previous to the examination. The pulse is most readily felt when the patient's forearm is pronated. In cases of aortic regurgitation the peculiar character of the pulse (*vide infra*, p. 189) is more distinctly brought out when the patient's arm is elevated.

To feel the pulse, place three fingers of the right hand on the patient's radial artery at the wrist. It is immaterial whether the observer's index finger be nearest the elbow or the hand of the patient, but for beginners it is best to select the same position in all cases. If it is made a habit to examine both radials in every case, errors in diagnosis, such as failing to detect the presence of aortic aneurysm or an abnormal position of the vessel, will frequently be avoided.

When the artery is beneath the finger, the following observations should be systematically made :—

(A) Factors which depend upon the heart.

1. Rate of pulse ;
2. Rhythm of pulse (irregularity, intermission) ;
3. Equality or inequality in strength of successive beats.

(B) Factors which depend upon the vessel.

4. The size of the artery ;
5. The condition of the vessel wall (*a*, hypertrophy of media ; *b*, changes in intima, *viz.* :—early, fibrous thickening ; late, atheroma).

(C) Factors which depend on both heart and vessels, the latter element generally being of greater importance.

6. The amount of movement during the passage of a pulse wave (volume) ;
7. The blood pressure in the vessel during the beat (maximum pressure or "force") ;
8. The blood pressure between the beats (minimum pressure or "tension") ;
9. The general character of the pulse wave as regards rise, maintenance, and fall of pressure ; and the presence or absence of palpable dicrotism or of other secondary waves.*

The **rate** of the pulse is given as so many beats per minute. It is well not to begin counting immediately the finger is laid on the pulse, as the agitation of the patient often accelerates it at first. After waiting till it resumes its normal rate, count the beats for one minute, and record the result. The beat at which the observation commences should not be counted.

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* In studying the pulse the student should always have before his mind the relation between the successive changes in pressure which can be felt with the finger, and the graphic record of these changes which can be secured by the use of the sphygmograph. The rise of pressure corresponds with the sphygmographic upstroke, the maintenance with the apex and portion of the curve following it as far as the dicrotic notch, the fall with that portion of the curve which commences with *the dicrotic wave* (*see* page 186).

The successive beats of the pulse may recur at equal or unequal intervals, giving a **regular or irregular rhythm.** In the latter case, the beats may occur in symmetrical groups, some of the simpler of which have received special names: thus if there be two beats and a pause, recurring in regular sequence, we obtain the *pulsus bigeminus* (Fig. 46), three beats and a pause give the *pulsus trigeminus* (Fig. 47). In other cases no such symmetry occurs. Besides the varying interval that may occur between consecutive beats, the beats themselves may be unequal in **strength.** Some beats may be weaker, and the weaker beats may eventually become imperceptible—we thus arrive at pulses which may be classed under the previous heading (irregular pulses); this is especially true of the symmetrical types.

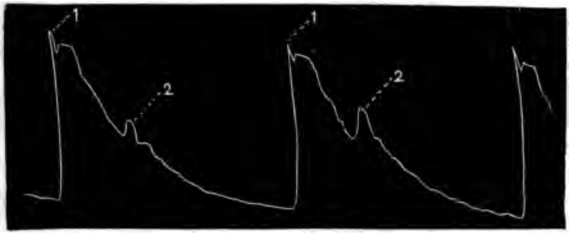

Fig. 46.—Bigeminal pulse. (*From a tracing lent by Dr. Byrom Bramwell, "Students' Guide to Examination of the Pulse."*)

The fourth observation is directed to ascertain the **state of the vessel.** Two points should be noted: first, the size (calibre) of the vessel; secondly, the *condition of its walls.*

To determine the *calibre*, empty the vessel of blood by firm pressure, and endeavour to gauge its breadth in the flattened state. Should this be impracticable, allow the blood to return below the finger, and note the size of the cylindrical tube. When the vessel is contracted the calibre is small, when the muscular coat is fully relaxed it is distinctly larger. Be careful to ascertain that the radial artery is really under observation; in a certain proportion of cases it winds round to the back at an unusually high level, whilst the superficialis volæ continues to run in the usual site of the larger vessel. One may thus be led into error if the abnormal distribution is not recognised.

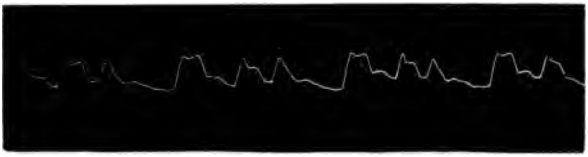

Fig. 47.—Trigeminal pulse.

To discover the *state of the walls*, flatten the vessel and cause the skin of the patient's wrist to slip up and down over it. In health the vessel wall can rarely be felt unless the arm is thin. In disease one may feel general or local thickening, calcification, tortuosity, or irregular dilatations. These changes must be discounted when an attempt is made to estimate the blood pressure in the vessel.

Having observed the state of the vessel, one proceeds to apply just sufficient pressure to flatten it between the beats. When this is done the increased blood pressure that is present during the beat will cause the blood vessel to resume its cylindrical shape; and further, inasmuch as the vessel wall is elastic, will stretch it until the internal stress is balanced by the strained wall.

This observation enables us to estimate the **amplitude of movement** of the vessel wall during the passage of the pulse-wave. As the elastic stretching of the vessel is never great, the movement chiefly depends on the resumption by the flattened artery of its cylindrical shape, and the amount of such movement is consequently greater the more dilated the vessel is. The force of the heart's action also exercises a certain influence on the amount of movement, although to a much less degree than the relaxation or contraction of the coats of the artery.

This proceeding is often described as "*observing the expansion*" of the pulse.

The next point is the determination of the **maximum blood pressure or force,** which, of course, occurs during the beat. In this case three fingers must be placed on the artery, so that it may be compressed both above and below the point where the pulse is being felt.

Place the finger next the wrist firmly on the vessel to prevent any pulse from the ulnar artery reaching the middle finger through the palmar arch; let the middle finger rest on the vessel with such pressure as will render the pulse most distinct, and then gradually compress the artery above this point with the remaining finger, noting the pressure employed when the pulse ceases to be felt by the middle finger. This pressure, being just sufficient to prevent the blood from lifting the finger during the beat, corresponds to the maximum blood pressure. Only careful practice on a large number of healthy and diseased pulses will enable the student to determine whether in a given case the requisite pressure is normal, excessive, or diminished.

The importance of cutting off recurrent pulsation through the palmar arch must never be overlooked. *It is most likely* to occur in cases where, owing to vascular dilatation, the blood pressure is unusually low, and thus if neglected would lead to serious error, as the middle finger would continue to feel the pulse, even when the upper finger exerted great pressure. As a natural consequence the observer would greatly over-estimate the maximum blood pressure in the vessel. It is worth noting that this recurrent pulse is frequently present in chlorosis.

It is rather more difficult to estimate the **minimum blood pressure, or tension.** One thing may be remembered—namely, that in cases where the maximum pressure is low the minimum must be still lower; this rule must not, however, be extended into an assertion that a high maximum will necessarily involve a high minimum pressure.

To test for the minimum pressure, one may attempt to roll the vessel from side to side under the fingers between the beats. When the pressure is low, one cannot feel the vessel at all; if it is high, it may feel as hard as a piece of whipcord. Of course, one must be careful not to be misled by thickening of the wall of the artery.

Another method which gives good results, and which may be more easily appreciated by many, is to feel the pulse first with light, then with moderate, and finally with considerable pressure of the fingers on the artery. A pulse of low tension (*i.e.* with a low minimum pressure) is best felt in the first case, for the light pressure is sufficient to flatten the vessel between the beats, whilst it allows the artery to resume its cylindrical shape without much resistance during the beat, and is thus favourable to the development of the greatest possible amplitude of movement; whilst on the contrary, where the tension is high, considerable pressure is required to flatten the *vessel between* the beats. But one obtains the greatest amplitude of movement precisely when the vessel is thus flattened, and so in a high-tension pulse the more firmly one presses the more forcible does the pulse appear to grow.

A normal pulse, lying as it does between these extremes, is best developed when moderate pressure is applied.

When all these points have been determined, one should conclude by studying the **general character of the pulse-beat.** This is divided into three periods: first, the period during which the blood pressure is rising; secondly, the period at which the blood pressure continues near its maximum; and lastly, the period during which the blood pressure once more falls off.

The amount of movement having been already observed, one tries to estimate the rapidity of the rise of pressure, describing the rise as abrupt, rapid, moderate, or slow. In cases where it is abrupt it will be found that the pulse is of low tension; where it is slow the pulse is either one of high tension or an aneurysm is present. The latter condition may cause the rise to be excessively gradual. It is only in aneurysm that the rise of pressure may be as gradual as the subsequent fall.

As regards the period at which the pressure remains near its maximum, the point to be observed is whether the pressure is well sustained, or whether, on the other hand it has no sooner attained its highest value than it begins to fall off again with rapidity.

In the third period, one observes whether the fall of pressure is swift or gradual. During the fall, instead of continuous decrease, there may be oscillations of pressure, which, in marked cases, are quite perceptible to the finger as distinct impacts following the primary stroke of the pulse. Of these the most *noteworthy* is that known as the **dicrotic wave.**

It is best marked in pulses of low tension (provided there is not aortic regurgitation), and is most readily felt when the finger is very lightly applied to the vessel. Where not readily perceived, it is often accentuated by occluding the vessel on the distal side by the lower finger, whilst the middle and upper ones remain lightly applied.

The secondary wave which the finger detects with the next greatest frequency is the **tidal or predicrotic wave.** It occurs in certain pulses of high tension where the blood escapes slowly from the contracting ventricle, and is very characteristically present in aortic stenosis. In opposition to the dicrotic wave, it is best felt when considerable pressure is applied to the artery, and thus there is no difficulty in discriminating between the two.

Besides these, in pulses of high tension, there are frequent fluctuations of pressure at a period subsequent to that in which the dicrotic wave occurs.

These can sometimes be detected by the fingers, but it requires much practice to recognise them. They are visible enough in good sphygmographic tracings.

The **typical pulse** of a healthy adult man should be described in some such terms as the following :—

The rate is seventy per minute. The beats are regular in rhythm and equal in strength. The expansion is moderate in amount. The vessel is not tortuous, its walls are not rigid or thickened, and between the beats it is just possible to feel it. It is of medium size. The passage of the pulse-wave can be arrested by moderately firm pressure, and the beat is best felt when a medium degree of pressure is applied : in other words, the force and tension are both moderate. With regard to the general character of the pulse, the rise of pressure is neither abrupt nor very gradual, it is fairly well sustained, and its fall is *gradual, but not* very tardy. No conspicuous oscillations of pressure are discernible during the time of subsidence.

The advantage of an instrument which could be used in clinical work for the accurate measurement of the arterial blood pressure both during and between the beats has long been recognised. We are now in possession of several which, though they cannot be wholly free from risks of error, since the pressures have to be gauged through the walls of the arteries, which are sometimes thick and rigid, and also through the tissues overlying them, are yet sufficiently trustworthy to offer a very material assistance in the estimation of blood-pressure, and, whilst supplementing and confirming observations which have been made by the finger, have the additional advantage of yielding their results in a form capable of being definitely recorded.

Of these instruments the most suitable for clinical use are the pocket form of Hill & Barnard's sphygmometer* and Oliver's hæmo-dynamometer.† In both of these the blood-pressure is estimated by the application of increasing pressure over the radial artery through a small bag containing fluid, the compressing force being estimated in Hill & Barnard's instrument by the height to which a column of fluid is driven up a closed tube against the resistance of the air confined above it. In Oliver's a similar method of determining the pressure is used, and its amount is read off on an ordinary pressure gauge, which has been carefully graduated.

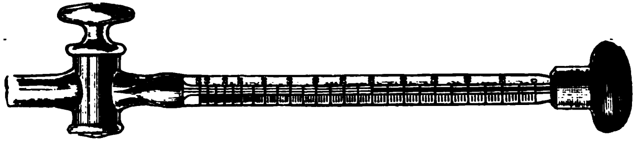

Fig. 48.—Sphygmometer, of Hill & Barnard (pocket form).

Hill & Barnard's instrument (Figs. 48, 48A) is used as follows:—

1. The arterial pressure should be taken when the patient, free from excitement, is quietly resting. It should be taken both in the recumbent and standing postures, in order to determine whether the compensation for the influence of gravity is perfect. The pulse frequency should be noted in each case. The wrist should be placed on the same level as the heart, and

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* Supplied by Hicks, Hatton Garden, London, E.C.

† *Supplied by Hawksley*, Oxford Street, London, W.

it is convenient to extend it over the edge of the bed or some other rest.

2. Hold the sphygmometer in the vertical position, open the tap, and press the edge of the rubber bag upon the table until the fluid rises to zero. Then close the tap. By setting the fluid at zero before each reading, errors due to variations of atmospheric pressure and temperature are avoided.

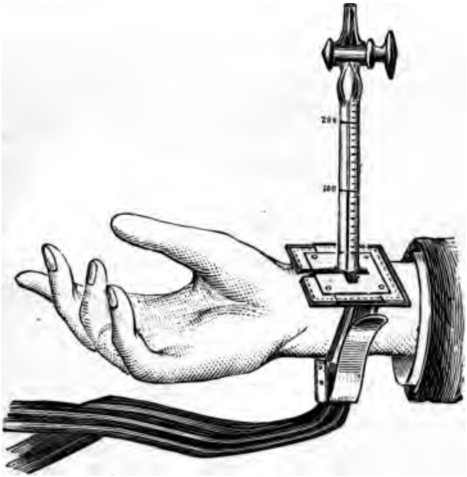

Fig. 48A.—Sphygmometer, of Hill & Barnard (pocket form, applied)

3. Should, however, the fluid have already risen above the zero mark, close the tap and press the fluid up until the chamber is about half-full, and on its return it will be found to have fallen below the zero mark, when you can proceed as stated above.

4. Having set the fluid at zero, press the rubber bag upon the radial artery until the maximal pulsation of the fluid is obtained. Read the pressures indicated on the scale at that point. For example:—Suppose the pulsation is maximal between 110 and 120 mm. Hg., then the arterial pressure equals 115 mm Hg. The normal pressure in young men is about *110-120 mm. Hg.* in the standing and 100-110 mm. Hg in the

recumbent posture. The pulse frequency does not, as a rule, vary more than five beats per minute in the two positions.

5. With this instrument the venous pressure can also be obtained in the manner suggested by Dr. George Oliver. On the back of the hand or arm choose a length of vein free from anastomosis, and press the sphygmometer upon the peripheral end of this. Then empty the vein centrally with the finger. Next, gradually reduce the pressure of the sphygmometer and note the exact pressure at which the vein refills with blood.

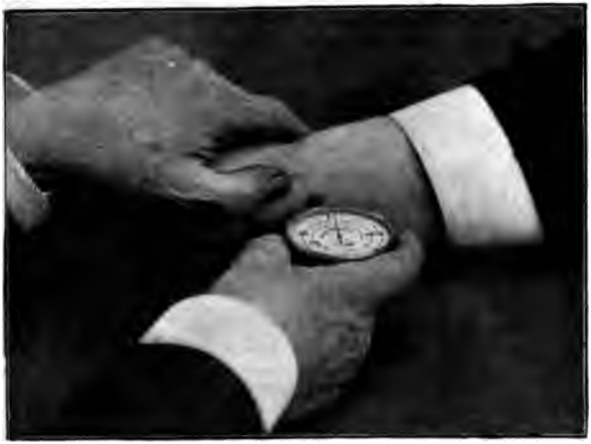

Fig. 49.—Oliver's Hæmo-dynamometer.

6. It is well to note that should the glass tap of the instrument become exceptionally stiff or tight, momentary suspension over the flame of a spirit lamp or candle renders it quite easy to turn again, but it is most important that it be held there no longer than is absolutely necessary.

The sphygmometer is also supplied with an attachment to fit the wrist, pressure being then increased or diminished by adjustments of the wrist band, and absolute steadiness thus ensured. (Fig. 48A.)

Oliver's instrument is employed thus :—

The wrist is bent at an angle of about 45°, so as to slightly *project the vessel on its* bed, while the arm rests on its ulnar side. The wrist should be on a level with the ensiform cartilage in the erect (sitting or standing) postures, and with the back in the recumbent attitude. The pad is placed over the artery whilst the instrument, with its dial uppermost, is pushed gently forward in the horizontal position (Fig. 49). The indicator will then rise on the scale, and begin to pulsate when measuring 50 mm. or more. The pulsations gradually increase in size as the indicator is made to rise further on the scale, until a point is reached when they attain their largest excursion, beyond which they as gradually diminish. The reading of the arterial pressure is made when the pulsations have acquired their fullest development; then the observer has raised the fluid pressure in the bag so that it just balances the blood pressure which is present in the artery between the beats, and its measure is the mean of the two readings at either end of the maximum excursion of the indicator. As it is more easy to determine with accuracy the limits of a small oscillation than those of a large one, it is better in cases where the volume of the pulse is large to select a smaller artery than the radial for the observation. In such cases the ulnar artery usually proves suitable.

Either instrument can also be employed to ascertain the maximum blood pressure by feeling the pulse beyond the point where the instrument is applied, and then gradually increasing the pressure until pulsation is no longer felt. The reading which the index then records is a measure of the maximum blood pressure in the vessel.

SECTION VII.—THE USE OF THE SPHYGMOGRAPH.

For permanent record, and also to aid in the analysis of details, it is important to employ a sphygmograph as well as to feel the pulse in all cases where the state of the heart and vessels is an important factor. Numerous types of sphygmograph are now in use, but only Marey's and Dudgeon's will be described here, as they are most frequently employed.*

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* A modification of Dudgeon's sphygmograph has been introduced by Jaquet, and has the advantage of possessing a time-marker actuated by a separate mechanism, which enables accurate measurements to be made of the exact duration of the various events *recorded in* the tracing. As the recording paper often

In **Marey's sphygmograph** the pad which rests on the artery acts on a long lever the farther end of which traces the pulse curve on smoked paper, that is held in a frame driven by clockwork. In **Dudgeon's** the same result is obtained by connecting two shorter levers in such a manner as to magnify the movement of the pad fifty times. The end of the lever in Marey's instrument describes the arc of a circle; in Dudgeon's, owing to the mechanical device employed, the style moves backwards and forwards in a straight line. The following description of the method of using each will aid the student in applying them:—

I. Marey's instrument: The patient should sit or lie down. Place the arm support on a table of suitable height beside the patient. Loosen any tight garments about the patient's arm, which must be bared to above the elbow. Place the arm supine on the support, with the back of the wrist at the highest part. Semiflex the fingers. Mark the line of the radial on the skin, and draw a cross line where the pad of the sphygmograph is to rest. Wind the clockwork and adjust the paper on the instrument.* Place the pad of the sphygmograph very accurately in position, with the lever pointing up the arm towards the elbow, and fix the instrument in its place by non-elastic bands. Adjust the pressure of the pad, by means of the screw or milled head, until the lever affords the maximum range of movement. Adjust the level of the style, after recoupling it with the pad,

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advances with considerable irregularity in speed this is an important improvement. A larger form of Jaquet's instrument is also made, in which by the aid of tambours the apex beat of the heart and the carotid pulse can be recorded simultaneously with the radial pulse.

* The paper is best smoked over a small piece of burning camphor, which may, if necessary, be moistened with a drop of alcohol.

so that the movement of the lever takes place opposite the smoked paper, and see that the style is in contact with, but does not press too heavily on, the latter. Before varnishing the trace, note patient's name, date (and time of day), whether right or left radial, pulse rate, respiration, and approximate pressure employed.* Ordinary quick-drying negative varnish, as used by photographers, is employed to fix the trace, the paper being dipped into a jar of varnish, or laid, trace upwards, in a saucer containing some.

II. Dudgeon's instrument :—

1. Wind up the clockwork.
2. Insert one end of the smoked paper (smoked side uppermost) on the right hand side of the instrument between the roller and the small wheels.
3. Make the patient hold out either hand open, and in an easy position, palm upwards, the fingers pointing towards you, and tell him not to move the wrist or fingers.
4. Ascertain and mark the precise spot where the radial artery beats at the wrist.
5. Slip the band, the free end of which has been drawn through the clamp, over the patient's hand.
6. Adjust the pressure of the spring.
7. Place the pad on the artery, the clockwork case being nearest the elbow.
8. Retain the instrument in place with the right hand ; tighten the band sufficiently with the left, and clamp by means of the screw with the right hand. When the band is correctly tightened, the needle will oscillate over the paper. If the tightness is nearly but not quite correct, bend the hand backwards at the wrist to increase the tension, or forwards to diminish it.

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* The nominal pressure as given by the index on the screw is not even approximately correct, but if the same instrument is always used the results are comparable to some extent.

9. Set the paper in motion by releasing the catch that controls the clockwork.

10. Let the paper run through unassisted, and catch it in your hand as it passes from the instrument. Generally the patient's hand must be supported whilst the tracing is taken.

11. Stop the clockwork as soon as the paper has passed.

In a pulse tracing, rise of blood pressure will be represented by an upstroke, and fall by a downstroke.

Bearing this in mind, the student will readily understand the main outlines of a healthy pulse tracing. The pressure rises fairly rapidly; therefore the upstroke, when the paper is driven forward at the usual speed, is nearly, but not quite, perpendicular. The percussion wave is quickly followed by what is known as the tidal (or predicrotic) wave; these are not separately distinguishable by the finger in health; the sphygmograph, however, indicates their existence by a notch at the beginning of the downstroke. Thereafter the pressure begins to fall off, but, at the moment when the aortic valves close, the decrease of pressure is arrested, and a positive (dicrotic) wave is propagated into the vessels: this condition is recorded by a small break in the descent of the downstroke of the tracing. The foot of the notch immediately before the dicrotic upstroke indicates the time when the aortic valves close. After this rise the line again curves downwards, often exhibiting slight secondary oscillations, until a new upstroke marks the arrival of the next pulse-wave. Ordinarily the blood pressure takes much longer to fall than to rise, hence the downstroke is much less vertical than the upstroke (Fig. 50).

A line joining the lowest points of the upstrokes of successive pulse-beats on the tracing is often known as the respiratory line. In health it is usually straight, but where the blood pressure is low and the patient is suffering from dyspnœa it becomes undulating, rising to a higher level during expiration and sinking again during the period of inspiration (Fig. 51).

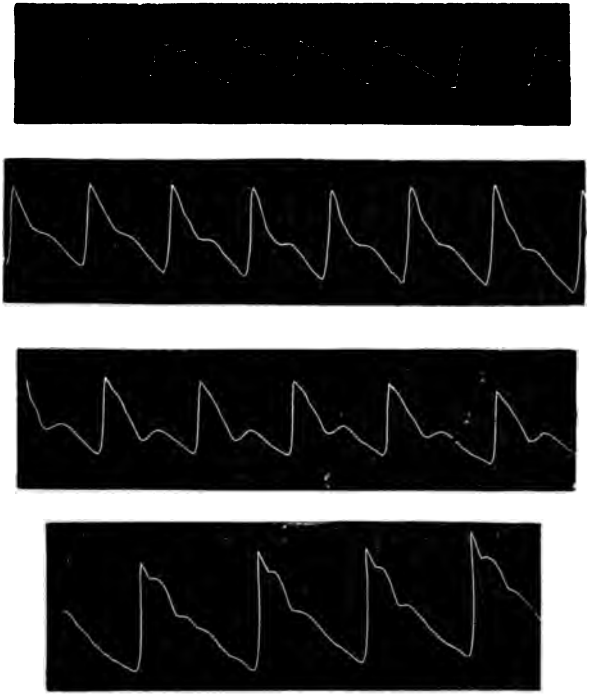

Fig. 50.—Normal forms of pulse. (*Mahomed.*)

In health a pulse tracing taken with suitable *pressure has a sharp* apex, a small tidal wave, and a moderately distinct dicrotic wave. A rounded apex, in most cases, means either excessive pressure of the spring or bad application of the instrument (*see*, however, p. 190).

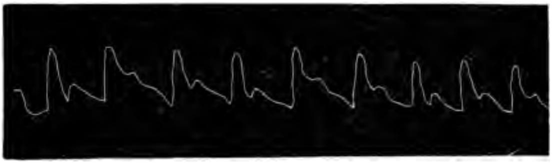

Fig. 51.—Low-tension pulse.

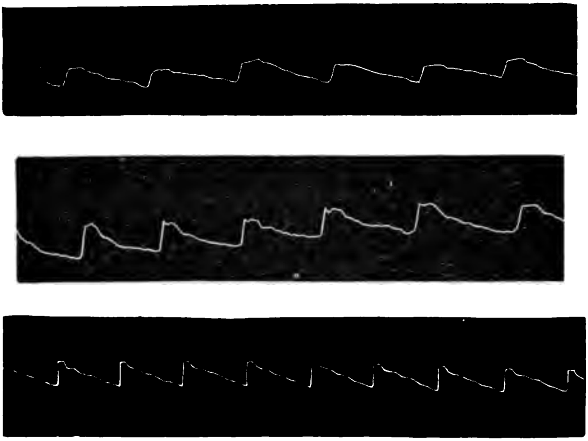

Fig. 52.—High-tension pulse.

The upstroke is longer and steeper than usual when the ventricle discharges a larger volume of blood than normal into the arteries, and when the *arterioles* are dilated (low tension) (Fig. 51).

The upstroke is shorter and less steep when the heart acts feebly or when the aortic ostium is stenosed, so that less blood than usual is delivered in a given time, and also when the blood pressure is high and undue opposition is thereby offered to the outflow from the heart (Fig. 52).

In conditions where the minimum blood pressure is low, the dicrotic wave is well marked; where it is high, the dicrotic wave is small, and secondary oscillations are present.

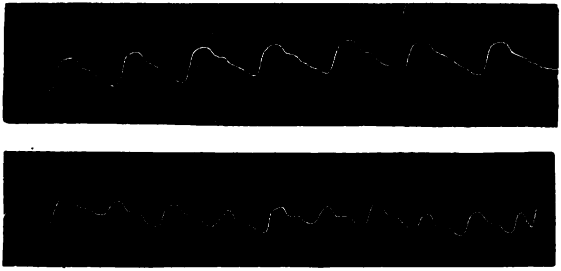

Fig. 53.—Aortic stenosis.

The following conditions give characteristic tracings:—

1. **Aortic stenosis.** Small amplitude, sloping upstroke, tidal wave well developed, and often higher than primary apex [anacrotic pulse] (Fig. 53).
2. **Aortic incompetence.** Great amplitude, abrupt upstroke, rapid fall, little or no dicrotic wave. This is known as the water-hammer, collapsing, or Corrigan pulse (Figs. 54, 55).
3. **Mitral disease.** Small amplitude, moderately steep upstroke, secondary waves rather slight, successive beats unequal and irregular, especially *when failure of heart* is threatening to set in. In

mitral stenosis the vascular tension tends to be higher than in mitral incompetence* (Fig. 56).

4. **Aneurysm** of the ascending or transverse aorta generally affects the pulse in the implicated radial, where the impulse is delayed, whilst the rise is gradual, the amplitude less, and the apex rounder than in the unaffected radial (Fig. 57).

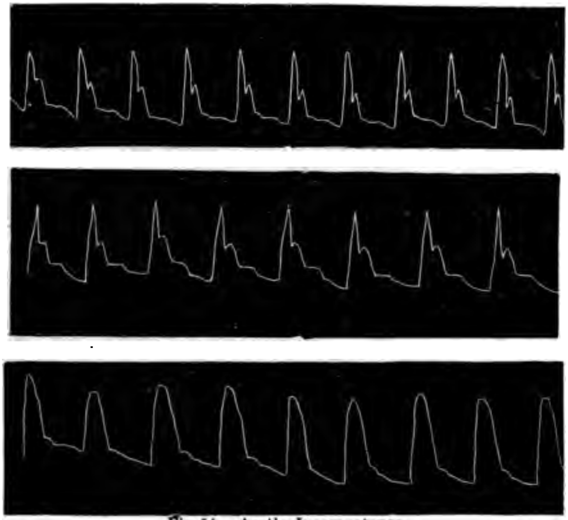

Fig 54.—Aortic Incompetence.

5. **Arterial atheroma** of the great vessels, by abolishing the modifying effect of their elasticity, renders the tracing similar to one taken from the pressure curve of the left ventricle. This is known

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* The characters of the pulse in mitral disease are very variable, and space forbids any complete analysis of the subject here. Those who wish to study the subject more fully are referred to Dr. Graham Steell's "Sphygmograph in Clinical *Medicine*," *p. 25*.

as the senile pulse (Fig. 58) Aortic atheroma combined with a moderate degree of incompetence gives a pulse which is like that of atheroma, but with sharper apex.

Several types of pulse have received special names; of these the following are amongst the more important:—

1. **Pulsus bisferiens.** The tidal wave is felt separately from the primary impact. The tracing is characteristic, and is often observed in cases of aortic stenosis (Fig. 59).
2. **Pulsus dicroticus.** The dicrotic wave is exaggerated (*see* p. 178) (Fig. 60).
3. **Pulsus anacroticus.** The tidal wave is more forcible than the percussion wave. Consequently the latter, instead of appearing at the summit of the sphygmographic tracing, produces an indentation in the upstroke of the curve, whose apex is formed by the tidal wave. The condition is found in pulses where the tension is high, and like the pulsus bisferiens is frequently associated with aortic stenosis. It is most important to distinguish from the true anacrotic pulse those cases where one finds that, from the rapidity of the heart's action and extreme low tension, the pulse exhibits *hyperdicrotism.*

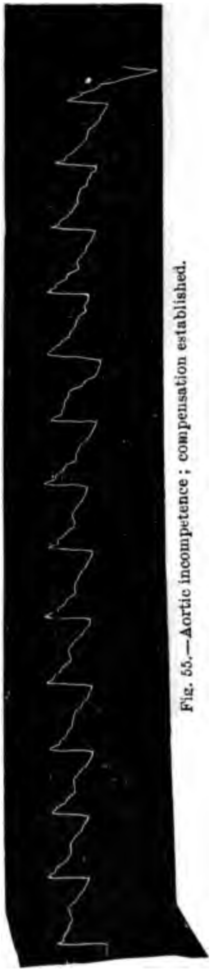


Fig. 55.—Aortic incompetence; compensation established.

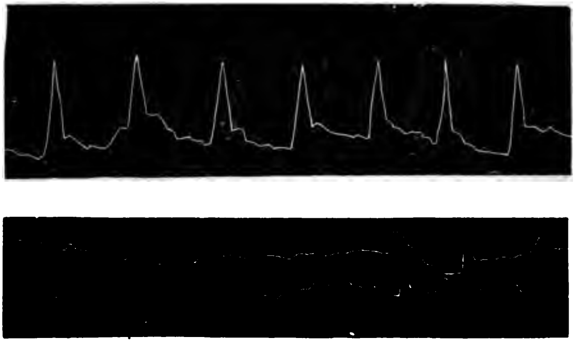
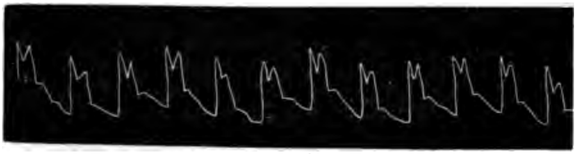


Fig. 56.—Mitral incompetence.

Left side.



Right side.
Fig. 57.—Pulse in aneurysm.

4. **Pulsus celer.** The pressure is ill sustained the up and down strokes are therefore abrupt.

5. **Pulsus tardus.** The pressure is well sustained, the tracing is less abrupt. This is sometimes described as a "long" pulse.

6. ***Pulsus bigeminus.*** There are two beats

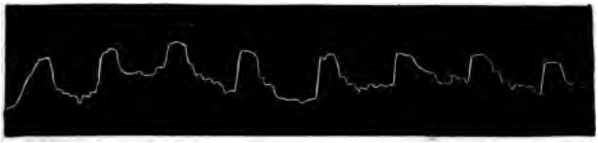


Fig. 58.—Aortic atheroma (senile pulse).

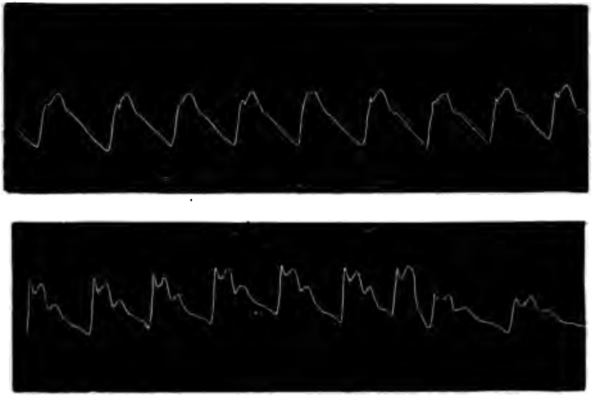


Fig. 59.—Pulsus bisferiens.

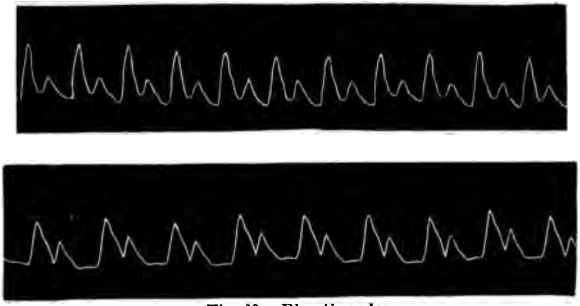


Fig. 60.—Dicrotic pulse.

and a pause. The two beats may be alike, or they may differ in force (Fig. 45).

7. **Pulsus trigeminus.** Three beats and a pause (Fig. 46).

8. **Pulsus paradoxus.** The pulse becomes smaller or disappears at the end of inspiration, when the patient breathes deeply. It occurs in pericardial adhesion.

9. The pulse is described as "**wiry**" when the vessels are contracted and the heart-beat is rapid and moderately strong. This may occur in peritonitis. When the heart gets weak, whilst the other conditions continue, the pulse grows "**thready,**" but at the same time the blood pressure generally begins to diminish.

10. The pulse is said to be "**running**" when the vessels are relaxed, and the heart's action is weak and fairly rapid.

SECTION VIII.—THE VENOUS PULSE.

In a considerable number of cases where the circulation of blood through the right side of the heart is interfered with, either from valvular disease or from increased blood pressure in the pulmonary circulation, the embarrassment manifests itself by distention or pulsation in the veins. From their size, nearness to the heart, and comparatively superficial situation, the veins of the neck offer special facilities for the study of these phenomena.

Whilst inspecting the root of the neck the observerhas already had an opportunity of noting these appearances where they exist, but the several varieties and degrees of disturbance in the venous circulation must now be more completely distinguished. A mere flicker of pulsation at the root of the neck when the patient is recumbent is common enough even in perfect health, and must not, in the absence of further evidence, be regarded as indicating any disease of the heart. When, however, the pulsation passes farther up, the case is entirely altered. Two points must then be noted—first, whether there is actual regurgitation, or merely retarded emptying of a full vein during the beat of the heart; and second, the exact moment at which the pulsation occurs with reference to the apex beat. Sometimes there is no difficulty in distinguishing between **regurgitation** and simple **undulation** in the vein. In bad cases of tricuspid incompetence, a mere glance at the patient will at once reveal the nature of the phenomenon. Where the conditions are less urgent, and the distinction is less readily determined, a simple plan is to empty the vein from below upwards by running the finger along it, and then to keep its upper extremity closed by the pressure of the finger, so that no blood can enter it from the periphery.

In cases of simple undulation, the vessel either remains empty and collapsed, or at most refills very gradually, as small collateral branches discharge their contents into it. Where, however, there is true pulsation, the valves at the root of the neck have ceased to be competent. As a consequence of this, coupled with the over-loaded state of the right heart, a backward wave of blood is forced into the vein, which re-fills from below with a series of bounds corresponding to the beats of the heart, and after three or four pulsations is again distended and pulsating as vigorously as ever, although the observer's finger continues to maintain the peripheral closure.

Regarding the time of occurrence of the venous pulse a great divergence of opinion has existed. Recent observation* leads to the following conclusions :—A large wave is, in typical cases, driven back during auricular systole; a short time after it has obtained its highest pressure, and when it is just beginning to fall off, it is reinforced by the shock which the carotid pulse imparts to all the adjacent structures. Thereafter the pressure again falls, and often reaches its minimum towards the middle of ventricular systole, when the obstacles to the entrance of blood into the auricle seem to be less marked.

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* *James Mackenzie*, "Pulsations in the Veins," Journ. of *Pathology and Bacteriology*, vol. i., p. 53. *Cf.* also Philipp Knoll

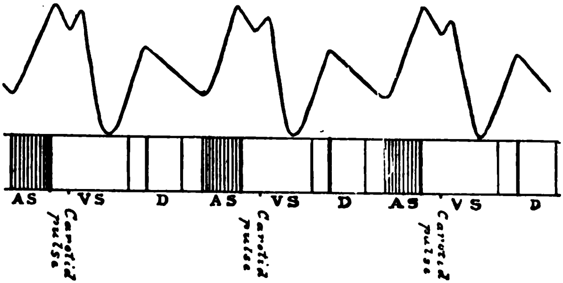

Fig. 61 (*see* text, p. 197).

The pressure again rises near the end of ventricular systole, the exact time of this second rise depending, on the one hand, on the amount of tricuspid leakage —or in some cases perhaps of mere backward yielding of the tricuspid curtains without leakage—and on the other, on the activity of the auricle; it then falls off somewhat during ventricular diastole, to rise once more at the commencement of the next cardiac cycle with auricular systole. These conditions may be traced in relation to the events of the cardiac cycle, when the sequence will resemble that of the accom-

"Ueber den Venenpuis," and "Ueber wellenförmige Blutdruck*schwankungen im* Venensystem," Pflüger's Archiv., Bd. 72, s. 317, 621.

panying diagram, where the upper tracing represents the movement of the recording lever of a suitably adjusted tambour applied to the pulsating vein, whilst the lower part indicates the events in the cardiac cycle, which correspond to the various parts of the curve so obtained (Fig. 61).

The details of individual tracings differ greatly; in some, one part of the curve, in others another part, being specially emphasised. For details the original paper must be consulted.

In certain circumstances a venous pulse of totally different origin may be present. This is known as the **centripetal venous pulse,** and is due either to great dilatation of the arterioles, so that the arterial pulse passes through the capillaries and is visible even in the veins, or to an aneurysmal varix, whereby direct communication occurs between an artery and vein, and the pulse-wave of the artery reaches the vein by this channel. The observer can have no great difficulty in recognising the nature of such centripetal pulsation.

SECTION IX.—SYMPTOMS AND SIGNS OF THE PRINCIPAL DISEASES OF THE HEART.

1. **Valvular diseases.**—(*a*) **Aortic incompetence.** The patient complains of attacks of giddiness, is often pale, and his arteries pulsate. Capillary pulsation may be observed. The apex beat is displaced downwards and outwards, and has a heaving character. The left border of the heart is farther out than usual. A murmur accompanies the second sound, as has been elsewhere described, and the pulse exhibits a "water-hammer" character.

(*b*) **Aortic stenosis.** Here the apex beat is *rather weak, and* the displacement less notable than in the last condition. The arteries are small, and the tension of the pulse, before compensation is destroyed, is somewhat high. Vertigo or fainting fits are not uncommon. The murmur is described on p. 163. The aortic second sound is very weak.

(*c*) **Mitral incompetence.** This condition is often associated with dyspnœa, which is the first symptom of which the patient may complain. The face is apt to be cyanotic. The apex beat is of moderate strength, and is frequently displaced outwards. On auscultation, in addition to the characteristic murmur, one finds marked accentuation of the second sound in the pulmonary area.

(*d*) **Mitral stenosis.** Here the stress falls chiefly on the left auricle and right side of the heart. The apex beat is therefore only slightly displaced outwards, and is not unduly vehement. The murmur is very frequently accompanied by a thrill, and in many cases the second sound as heard at the apex is reduplicated. The pulse is at first not notably affected, but gradually becomes irregular and feeble as compensation fails.

(*e*) **Tricuspid incompetence,** which is usually secondary to mitral disease, is associated with dilatation of the right auricle, and consequently with outward displacement of the right border of the heart, with venous and hepatic pulsation, and with dropsy. The murmur has already been described; the second sound in the pulmonary area is weak. The patient usually suffers from very decided dyspnœa.

2. **Idiopathic heart diseases** lead at first to hypertrophy and dilatation of the ventricles, subsequently to fatty changes in the myocardium. The physical signs will obviously vary with the stage of the disease, and are frequently somewhat hard to interpret. Like all other forms of heart disease,

they terminate in failure of compensation, with its accompaniments of dyspnœa, urinary deficiency, dropsy, and cyanosis.

3. **Pericarditis** is characterised by pain in the chest, more or less fever, and by friction of a to-and-fro character when effusion is scanty. When more fluid has been poured out, the area of dulness is triangular and oversteps the second left interspace. The first indication of an increase in the area of dulness is often found to the right of the sternum immediately above the point where the liver and heart dulness meet. The apex beat is internal to the left limit of dulness, and may be weak or quite unable to be felt. The pulse may be greatly affected, and it is by this disease that the "pulsus paradoxus" is produced.

4. **Intrathoracic aneurysm** of the aorta leads in some cases to a tumour at the upper part of the chest, which is dull on percussion, and in which expansile pulsation can often be observed. It may also affect the character and synchronism of the pulses at the two wrists. The auscultatory phenomena are very variable. It produces numerous symptoms by involvement of different structures upon which it comes to press as it enlarges.

5. **Cardiac asthma.** This condition is characterised by attacks of severe dyspnœa, often lasting for several hours on end. The patient is so breathless that he requires to sit up, and may have to call the accessory respiratory muscles into violent exercise. It is distinguished from bronchial asthma by the state of the heart, which is generally dilated, and whose apex beat is weak. The pulse also is small, rapid, and irregular, and the dyspnœa is not of the pure expiratory type which characterises asthma of respiratory origin.

CHAPTER V.

CLINICAL EXAMINATION OF THE BLOOD.

Enumeration of red blood corpuscles.—This may be done by means of either a Thoma-Zeiss or a Gowers' hæmocytometer. The former is the simpler and more accurate instrument. We shall describe it first.

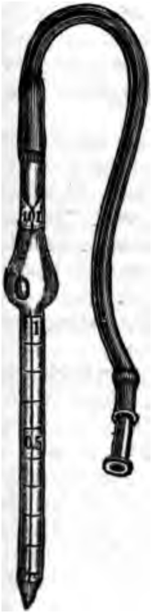

Fig. 62.—Hæmocytometer pipette (Thoma-Zeiss).

1. **Thoma-Zeiss hæmocytometer.**—The instrument consists of a mixing pipette (Fig. 62) suitably graduated and a counting slide. Cleanse the lobe of the patient's ear with soap and water, dry it, and rub it a little between the finger and thumb in the drying, so as to render it hyperæmic. Make a puncture on the lower border of the lobe by means of the lancet-shaped needle supplied with the instrument. The needle should be inserted with a rather sudden stab—not too slowly—and the blood must flow freely. On no account must the blood be *squeezed* out, as it is then always diluted by lymph squeezed out of the tissues. Slowly suck up blood by means of the pipette till either the mark 0·5 or 1 is reached. If one should happen to go a little beyond the 0·5 mark, the column of blood should be gently blown down to the proper point. If blood has been sucked past the mark 1, it has reached the

mixing chamber, and the process must be begun over again. Having charged the pipette, wipe the end of it on a clean cloth, and plunge it at once into the diluting fluid (Appendix, 17), which should be standing ready in a small, wide-necked bottle with the stopper out. Suck up the diluting fluid as far as the mark 101. Whilst doing so, the pipette should be gently rotated so as to start the mixing. Seize the pipette firmly by its ends between the forefinger and thumb, and shake thoroughly for about one minute. This produces a thorough mixing of the blood with the fluid. It must be remembered that the column of diluting fluid which occupies the capillary part of the pipette does not enter into the mixture. Hence, if blood is sucked up to 0·5, the dilution produced is in the proportion of 1 in 200, whereas if blood is taken up to the mark 1, the dilution is only 1 in 100. The former degree of dilution is to be preferred in most cases. The finger should now be removed from the pipette, and the diluting fluid in the capillary tube blown out. After a few drops of the diluted blood have been shaken out, a small drop is transferred to the counting slide (Fig. 63). The latter consists of a small platform (*m*) bounded by a trench which is surrounded by a glass slab (*c*). On the surface of the platform microscopic squares are ruled, each having an area of $\frac{1}{400}$ square millimetre. Special cover glasses, carefully ground, are supplied, which rest upon the glass slab, a space being left between the under-surface of the cover and the surface of the platform, which *space is* exactly $\frac{1}{10}$ millimetre in depth.

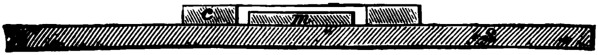

Fig. 63.—Thoma-Zeiss Counting Slide.
s, slide *m*, platform; *c*, wall of trench.

The drop of diluted blood should be placed in the centre of the platform, and should be of such a size that when the cover glass is placed in position the drop is flattened out so as to cover most of the surface of the platform, *but yet without any of it flowing over the edge into the trench.* It requires a little experience to enable one to take just the proper size of drop. It is important that the cover glass should lie quite flat upon the glass slab. This can best be achieved by previously washing both it and the slab with caustic potash, so as to remove all grease, and then rubbing them with soft chamois leather. The cover must be lowered into position by means of a needle. One recognises that the cover glass is lying properly by the appearance of concentric colour (Newtonian) rings between it and the slab. The rings should be visible when the cover is simply *lying* on the slab without any pressure being exerted. The rings are best seen by looking horizontally along the surface of the cover. If they are not visible at first, gentle pressure on the cover glass often brings them out. Unless the rings are seen, one cannot be sure that the space between the cover and the platform is exactly $\frac{1}{10}$ millimetre in depth. Having placed the drop in position, and the rings being visible, one should set the preparation aside for five minutes or so, to enable the corpuscles to settle. It should then be examined with the low power to see whether any air bubbles or foreign bodies are present, and whether the corpuscles are distributed with fair uniformity throughout the field, after which the high power is used for counting. The little squares will be seen to be marked off into sets of sixteen by double ruling (Fig. 64). Should the lines marking off the squares be only dimly seen, it may be necessary to intensify them. This is best done by rubbing the surface of the platform with a little finely powdered graphite—*e.g.* the scrapings from a very soft lead pencil—and then polishing it with soft chamois leather. If the microscope has a substage condenser the lines appear more clearly if it is racked down to some distance below the level of the stage.

For enumeration of the red cells, at least three sets of sixteen squares should be counted. The squares in each set should be gone over systematically in horizontal rows of four at a time. Of the corpuscles which lie *upon* the lines bounding the row, only those on the upper and on the left hand lines should be counted.

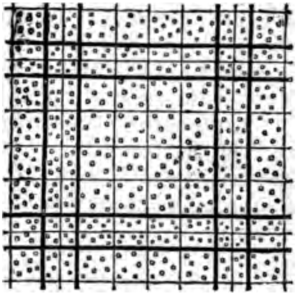

Fig. 64.—Microscopic View of Thoma-Zeiss counting-slide showing divisions.

Calculation.—Count the corpuscles in each of the four horizontal rows from above downwards. The total is the number of corpuscles in sixteen squares. Count in this way three sets of sixteen, and divide the total by forty-eight, which gives the average of corpuscles in one square. But the dimensions of this square are $\frac{1}{400} \times \frac{1}{10} = \frac{1}{4,000}$ cubic millimetre. Therefore, if there be x corpuscles in this dimension, there will be 4,000 x in 1 cmm. But the blood was diluted 200 (or 100) times. Therefore, in 1 cmm. of blood there will be $4,000x \times 200$ (or 100) corpuscles.

Suppose, for example, that one finds a total of 288 corpuscles in the forty-eight squares. This gives an average of six corpuscles per square, or 6 × 4,000 —*i.e.* 24,000 per cmm. of *diluted* blood, or 4,800,000 per cmm. of pure blood, if the dilution was one in *two hundred.*

The constant error in the Thoma-Zeiss instrument is less than 1 per cent. of the total result. The variable error depends upon the number of corpuscles counted. By counting 200 corpuscles, it amounts to 5 per cent. of the total; by counting 5,000 it amounts to only 1 per cent. To count the whole 256 squares takes about half an hour. This usually means counting 1,200–1,500 corpuscles. The co-efficient of error is then about 2 per cent.

2. **Gowers' hæmocytometer.**

The instrument consists of a capillary tube graduated for 5 cmm., and a pipette graduated for 995 cmm., a mixing vessel and stirrer, and a counting-slide.

Take up 995 cmm. of diluting fluid (Appendix, 17) in the pipette, and transfer it to the mixing vessel. Suck up 5 cmm. of blood in the capillary tube and gently blow it into the above quantity of fluid. Mix the two very thoroughly by rotating the stirrer between the finger and thumb. The blood is now diluted in the proportion of 1 in 200. Transfer a drop of the mixture to the centre of the ruled area on the counting-slide, cover, and fix the cover glass with the clips. The ruled area is divided into squares of $\frac{1}{10}$ mm. square, and the space between the surface of a square and the under surface of the cover glass is $\frac{1}{5}$ mm. in depth. Count the corpuscles in ten squares. The result multiplied by 10,000 is the number of red cells in a cubic millimetre of undiluted blood.

The normal **number of red corpuscles** is 5,000,000 per cmm. In some full-blooded adults this number may be exceeded. In females, even in health, it is usually rather smaller (about 4,500,000). The blood of newly-born children sometimes contains more than 5,000,000, and that number is also surpassed in cases of prolonged cyanosis.

The number is reduced in all forms of anæmia.

In chlorosis, however, the number of corpuscles may be normal, or nearly so.

Enumeration of leucocytes.

A special pipette is supplied for this purpose with the Thoma-Zeiss instrument. It is used in precisely the same manner as the red-corpuscle pipette, but permits of a lesser degree of dilution of the blood. The best diluting fluid to employ is 0·3 per cent. solution of acetic acid, to which enough of a watery solution of methyl green has been added to give the mixture a decided green colour. The advantage of this mixture is that it dissolves all the red cells while it stains the nuclei of the white. One can thus easily count the whites, and at the same time note the relative numbers of the uni- and multi-partite nucleated varieties.

It is important that a large drop of blood should be allowed to exude before one begins to fill the pipette. The blood should be sucked up to the mark 0·5, the end of the pipette wiped, and diluting fluid taken up to the mark 11.

Owing to the relatively large calibre of the pipette, the blood is apt to run out of it. It is well, therefore, to keep the pipette in a horizontal position so soon as one has filled it with blood.

The blood and the fluid are mixed as already described. This produces a dilution of 1 in 20. A drop is then placed on the counting-slide with the same precautions that were observed in the case of the red cells.

In this case the whole sixteen sets of sixteen squares should be counted, or 256 squares in all. Instead of going over the squares in rows of four, a whole set of sixteen can easily be counted at one time. A movable stage greatly facilitates the enumeration.

One should note on a piece of paper the number of *leucocytes with* multipartite and with rounded nuclei respectively in each set of sixteen squares. In this way one gets a rough idea of the proportion of each variety present, and by adding them together the total number of white corpuscles is obtained. The *calculation* is made in the same way as that of the red corpuscles, it being borne in mind that each of the 256 squares counted represents $\frac{1}{4000}$ cmm. of diluted blood, and that the dilution is much less than in the enumeration of the reds (1 in 10, or 1 in 20). For example, if there be twenty-five leucocytes in the 256 squares, this represents an average of $\frac{25}{256}$ per square, or $\frac{25}{256} \times 4,000$ per cmm. of diluted blood, or 7,812 per cmm. of pure blood if the dilution is 1 in 20. This is about the normal number.

In leucocythæmia, where a very large excess of leucocytes is present, one can easily count the red and the white cells in the same drop. For this purpose a 3 per cent. solution of common salt just coloured with a gentian violet is to be preferred for diluting the blood. This stains the nuclei of the whites, and at the same time preserves the reds. The dilution and calculation are the same as for the red cells.

In most cases, however, it is not easy to make an accurate estimation of the leucocytes by means of an ordinary hæmocytometer such as that of Gowers. Should its use be unavoidable, it will be found that the easiest way to pick out the white corpuscles from the mass of reds which tends to conceal them is to raise the microscope a little and then focus slowly down upon the field. The white corpuscles, being highly refractile, come into view first as little bright spots, and can thus be more easily enumerated. If Gowers' instrument is used, one must count the leucocytes in fifty squares, and multiply the result by 2,000. This gives the number of leucocytes in *1 cmm. of pure blood.*

The **number of leucocytes** in normal blood is about 7,000 per cmm., or 1 to 700 reds. The number varies, however, within considerable limits even in health. The normal proportion of leucocytes with divided nuclei to those with rounded nuclei is about 2 to 1.

A *physiological leucocytosis*, in which the small cells with rounded nuclei (lymphocytes) are both absolutely and relatively increased, occurs in infancy and after meals. In the *pathological leucocytosis*, met with in fevers and other conditions, the increase affects chiefly the cells with multipartite nuclei, and these may come to be ten or more times as numerous as the others. The condition of the leucocytes in *leucocythæmia* will be referred to later.

After use, the diluting pipettes should be thoroughly cleaned. A little trouble in this is repaid by saving of time and annoyance when next they come to be used. They should be washed out (1) with distilled water, (2) with absolute alcohol, and (3) with ether. A stream of air should then be blown through till one is sure that the glass ball in the chamber moves freely without tending to adhere to the sides. To save time in these manipulations, the rubber tube may be taken off and the fluid blown out through the wide end of the pipette. Coagulated blood may be removed from the capillary tube by means of a horse-hair. If the blood adheres firmly to the pipette, it may be removed by repeated rinsing with strong alkali or acid, or it may even require to be digested away with pepsin.

Estimation of blood platelets.

For this purpose the use of a pipette is not to be recommended, as the platelets adhere to its wall. One also requires to employ a diluting fluid of a certain degree of viscidity, otherwise the platelets are apt to run into groups. The best method of procedure is as follows :—

Place upon a slide a drop of a mixture of equal parts of glycerine which has been saturated with dahlia and 2 per cent. salt solution. The use of the dahlia is to stain the platelets. Touch the drop of blood with this mixture and cover. Count with $\frac{1}{12}$ lens in a succession of fields (1) the platelets, (2) the red corpuscles. Continue until 400 of the latter have been enumerated, and estimate the proportion of platelets to red corpuscles. Normally this should be as 1 : 8½. By then making an estimation of the reds in the ordinary way, one arrives at the number of platelets per cmm. Normally this is about 640,000. Variations in their number are not yet of any known clinical significance.

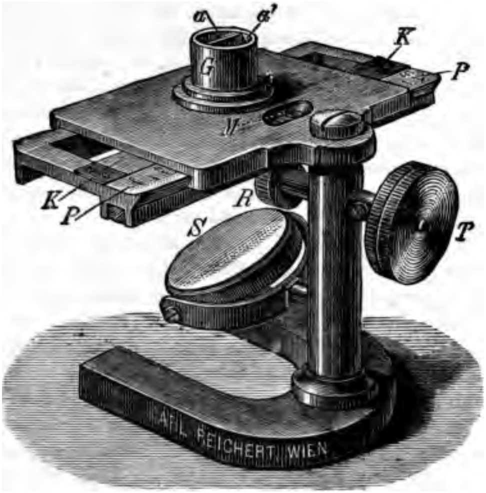

Fig. 65.—Von Fleischl's Hæmometer.

a', compartment above tinted wedge; *a*, compartment for blood; *K*, wedge turned by *T*; *P*, scale; *G*, cylinder; *M*, indicator; *S*, disc; *R*, screw for adjusting scale

Two precautions are necessary in making the above *estimation: (1)* To avoid *squeezing* the blood out of the puncture, and (2) to reject any preparation in which the platelets are found to have run together into clumps.

For the estimation of the amount of hæmoglobin one has the choice of several instruments:—

(1) **Von Fleischl's hæmometer.***—(Fig. 65). The instrument consists of a stage, resembling that of a microscope, with an aperture in the centre. Into this aperture there fits a short cylinder (G) with a glass floor. The cylinder is divided into two compartments (*a* and *a*′) by a vertical partition. Below the stage there is a frame carrying a wedge-shaped piece of coloured glass (K), which lies opposite the bottom of one half of the cylinder, and can be moved with a screw so that any thickness of the wedge can be brought below the cylinder. Light from a lamp (daylight will not do) is reflected into the bottom of the cylinder by means of a white disc (S). A small capillary tube attached to a wire is supplied with the instrument.

Method of use. Fill one compartment (*a*′) about a quarter full of distilled water. See that the capillary tube is quite clean and dry. To ensure this, one may, if necessary, pass through it a threaded needle, the thread being soaked in alcohol. Prick the lobe of the ear and insert one end of the tube sideways into the drop of blood. The tube fills itself, and must then be wiped on the outside so that no blood adheres to it. Plunge the tube into the compartment containing distilled water and shake it about by means of the wire handle till all the blood is washed out. If necessary, one may aid this by forcing water through the tube from a dropping pipette. The mixing of the blood with the distilled water may be completed with the aid of the wire handle of the tube, care being taken that no blood is allowed to lurk in the corners. One now fills up the rest of this compartment and the whole of the other with distilled water from a dropper. All daylight is now excluded, and then, looking down upon the cylinder, one turns the screw until a sufficient thickness of the coloured wedge has been brought under the other half of the cylinder to match exactly the tint of the blood. One then reads the figure on the scale (P), and this gives the percentage of hæmoglobin. The instrument is so constructed that when the screw is opposite the mark 100 a sufficient thickness of the coloured wedge has been introduced to match exactly the colour produced in the other half of the cylinder by the amount of blood which the capillary tube takes up, provided the blood contains a normal amount of hæmoglobin. To facilitate accurate matching of the two halves of the cylinder, the following precautions are necessary (Cabot):—

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* The best form of the instrument is that modified by *Mischer and made by* Reichert.

1. Use very little light. The less the percentage of hæmoglobin the less light one should use.
2. Look at the two halves of the cylinder through a tube made of a roll of black paper.
3. Do not sit opposite the light as one does when using a microscope, but sit at one side of the instrument. The light from the two halves of the cylinder then falls on the right and left halves of the retina, not on its upper and lower parts, the sensitiveness of which to colours is unequal.
4. Use one eye, and only look for a very few seconds at a time, since the colour sense soon gets fatigued.
5. Move the screw with short, quick turns, not gradually. Sudden changes in tint are more easily appreciated than those brought about gradually.

(2) **Gowers' hæmoglobinometer.** — Place a couple of drops or so of distilled water in the little graduated test tube supplied with the instrument. Get a large drop of blood from the ear, and fill the pipette with it up to the mark. Then dip the end of the pipette into the distilled water in the tube and gently blow out the contained blood. Mix and go on adding water drop by drop, comparing the colour from time to time with that of the standard tube. The latter is filled with tinted gelatine, and represents the colour of blood which contains a normal amount of hæmoglobin when diluted in the proportions effected by the instrument. The comparison should be made both by transmitted light, care being taken to hold both tubes level with the eye, and also by reflected light, the tubes being held side by side against a sheet of paper. Good daylight is indispensable. Stop adding water *when the tint in the two* is the same, and read off the

level at which the mixture stands in the graduated tube. If this be at (say) 60, then the blood contains 60 per cent. of hæmoglobin The mean of the dilution which is just too much and that which is just too little is the correct point.

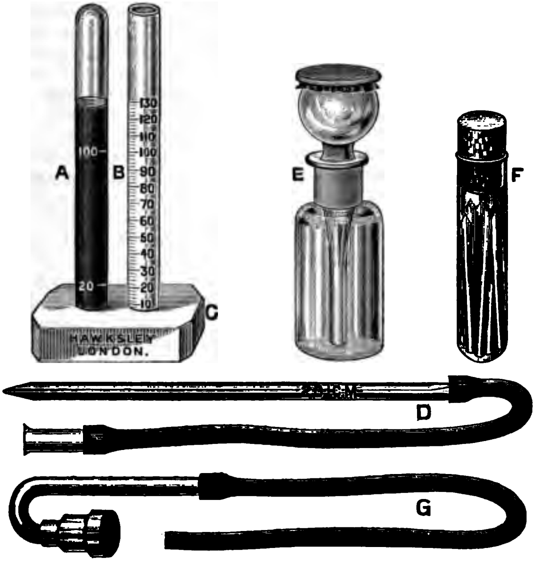

Fig. 66.—Haldane's Hæmoglobinometer.

A, standard blood solution ; B, graduated mixing tube ; C, rubber stand ; D pipette ; E, bottle for distilled water ; F, lancets ; G, tube and cap for fixation to gas burner.

(3) **Haldane's hæmoglobinometer.**—Haldane has modified Gowers' instrument by using as a standard of comparison, instead of gelatine tinted with picrocarmine, a 1 per cent. solution of blood containing the average percentage of hæmoglobin found in the blood of healthy men, and saturated with

carbonic oxide. It has an oxygen capacity of 18·5 per cent. as determined by the ferricyanide method, and is both definite and permanent.

The instrument is shown in Fig. 66. It is used as follows:—

Sufficient water is first placed in the graduated tube to dilute the blood as far as safely possible. A puncture is then made in a finger or the lobe of an ear, and the capillary pipette (which must be clean and dry) at once filled to a little beyond the mark 20 from the drop of blood obtained. The point of the pipette is wiped, and dabbed on any convenient surface until the contained blood stands exactly at the mark. The blood is then gently blown out into the graduated tube, where it sinks; the pipette is rinsed with the water in the graduated tube and withdrawn. The piece of rubber tube attached to a gas burner is now introduced into the graduated tube to near the level of the water, and gas allowed to pass for a few seconds. As the tube is withdrawn (with the gas still passing) the end is closed with the forefinger, and the liquid made to pass up and down in the tube—not violently shaken—at least a dozen times, so as to saturate the hæmoglobin with CO. During this manipulation the tube is held in a handkerchief, otherwise it will become heated and liquid will spurt out when the finger is withdrawn. Water is now added drop by drop with the pipette stopper, the tube after each addition being inverted, until the point is reached at which the tints of the liquids in the two tubes are just equal. In judging of the equality the tubes should be held against the light from the sky, or, if artificial light be used, from an opal glass shade. It is also absolutely necessary to transpose the tubes repeatedly; otherwise serious errors may arise. The level is read off on the graduated tube after half a minute has elapsed since the last drop added was mixed with the rest of the liquid by inverting the tube. The observation is repeated after the addition of another drop of water, and if necessary another, until the point is reached when the tints are again unequal. The true result is the mean of the readings giving equality. The error in any single determination ought not to exceed 1 per cent.

The result obtained is the percentage actually present of the average proportion of hæmoglobin in the blood of healthy adult men. The blood of healthy men, however, contains more hæmoglobin on an average than the blood of healthy *women* or children. Women give an average of 89 per cent., *and children* of 87 per cent. of the proportion in men. The results may be expressed in terms of oxygen capacity (the number of volumes of oxygen taken up in combination from air by 100 volumes of blood) if it be borne in mind that 100 per cent. on the scale corresponds to an oxygen capacity of 18·5.

The advantages of the modifications introduced into the original method of Gowers are: (1) that the standard solution is a definite one, so that an instrument can be verified at any time by making a determination with ox-blood of which the oxygen capacity has been determined by the ferricyanide method; (2) that the standard solution is permanent; (3) that the apparatus can be used with equal correctness by daylight and artificial light.*

(4) **Oliver's Hæmoglobinometer** (Fig. 67).—The apparatus adopted by Dr. Oliver is founded on the colorimetric principle. But the applications of that principle are modified in the following ways:—

(1) Double transmission of light (or reflected light) is used instead of single transmission.

(2) A standard white background is selected on which the solution of the blood and the standard colours rest.

(3) The standard is presented as a series of definite gradations.

(4) The colour of the blood solution is compared with that of the standard in camera.

The *apparatus* consists of an automatic blood measurer, a mixing pipette, the blood cell and cover glass, the sets of standard gradations, the riders, the camera tube, the light, a bottle of antiseptic fluid, the lancet, needles and thread.

The automatic blood measurer (*c*) has a capacity of 5 cmm. and fills readily by capillary attraction. The handle is useful for stirring together the blood and water in the blood cell.

The mixing pipette (*d*) is provided with a rubber nozzle which fits over the polished end of the blood measurer and ensures the complete rinsing out of the blood with the first few drops of water.

The blood cell (*e*) is of more than sufficient capacity to ensure

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* If the capillary pipette becomes blocked or soiled by coagulated fibrin, use the brass wire in the case from the wide end of the pipette, or a horse-tail hair, to remove it. (On no account use iron or steel wire.) To dry the pipette, suck air through it. Always examine the pointed end of the pipette before using it; *it should be smooth and rounded, if sharp or angular it is broken.*

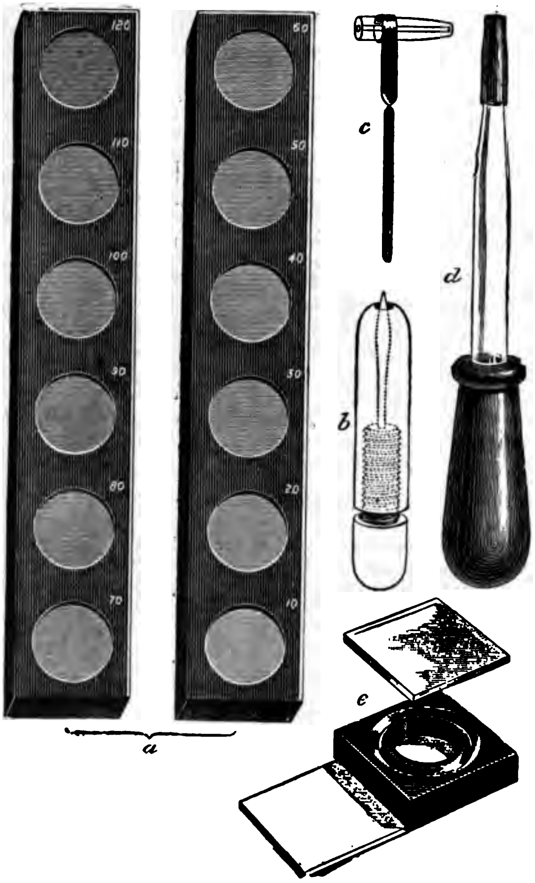

Fig. 67.—Oliver's Hæmoglobinometer (*see* text).

the complete liberation of the hæmoglobin. It yields a blood solution of rather less than 1 per cent. when filled level with the rim. It is itself the measure of the amount of water to be added, and it is quite easy to fill it accurately.

The standard gradations (*a*) are arranged as circular discs in two slabs—six in each; and they represent the divisions of 10 degrees of the scale from 10 to 120 inclusive.

The riders are small squares of tinted glass provided for the reading of the degrees intervening between each of the standard gradations.

The daylight standard is less adapted to the finer readings than the candle-light one, because the value of each rider when used with it is doubled, *i.e.* number one rider becoming equal to two degrees in the upper half of the scale and four degrees in the lower half; therefore the candle-light standard is preferable for such readings; and when it is used each rider has an equivalent value in the six stronger grades = 1 degree, and a double value in the six weaker grades = 2 degrees.

The light.—For observation by artificial light it has been found that the small-sized wax candles known as Christmas candles are the most satisfactory in affording the most suitable intensity of light, and one, moreover, which is sufficiently uniform.

How to make an observation.—The bore of the blood-measurer is first of all dried out by passing through it a needle threaded with darning cotton, and then the polished point is presented to the drop of blood. Care must be taken to see that the pipette is really quite filled, and, if it has been necessary to re-apply it to the drop, it should be observed whether there is a break in the column of blood. The rubber nozzle of the mixing pipette, charged with water, is adjusted over the polished end of the pipette, and the blood is washed into the blood-cell by pressing through the water drop by drop. The handle of the pipette is then used as a stirrer, and the further additions of water are made to impinge on it, for it serves to graduate the size of the drops required to accurately fill the cell. A final thorough mixing with the handle will be required, and perhaps another slight addition of water may be necessary to secure a level filling. The cover glass is then adjusted, when a small bubble should form, a sure sign that the cell has not been overfilled. The blood cell is then placed by the side of the standard gradations, and the eye quickly recognises its approximate position on the scale. Then the camera tube will more accurately define it. If it is found that the blood solution is matched in depth of colour by one of the standard grades, the observation is at an end, but if it is observed to be higher than one gradation but lower than that above it, the blood-cell is placed opposite to the former and the riders are added to complete the estimation.

In all tintometric observations take a standard time for looking down the camera tube (a ten-seconds observation is most convenient).

One can also state the percentage of hæmoglobin in terms of the amount contained in each corpuscle. Thus, if the number of red cells be 20 per cent. of the normal and the hæmoglobin 10 per cent., then the hæmoglobin value of each corpuscle is $\frac{10}{20}$ or $\frac{1}{2}$ normal. The importance of this method of expressing the facts is seen when one recollects that the total amount of hæmoglobin in the blood may be diminished while the amount in each corpuscle is really above the normal. This happens in some forms of anæmia.

The last point to be remembered in making all blood estimations is that, as far as possible, all observation on the same individual should be carried out under the same conditions as regards time of day, taking of food, etc. This is important, as it is found that the composition of the blood is temporarily altered by the taking of food, or by the occurrence of profuse sweating, diarrhœa, etc.

MICROSCOPICAL EXAMINATION OF BLOOD.

Blood may be examined (1) fresh, (2) fixed but unstained, (3) stained.

1. **Blood examined fresh.**—A drop of blood is transferred from the ear to a slide, covered, ringed with vaseline, and examined at once.

In the case of normal blood, the red corpuscles will be observed to arrange themselves in rouleaux as one watches, clear spaces being left between in which the white cells and little clumps of aggregated platelets may be seen. Any abnormality in the shape of the red cells or in the formation of rouleaux *should be noted.* One can also see if any large excess of white corpuscles is present. The presence of abnormal elements should be noted. Among these are abnormal varieties of white cells, more easily recognised, however, in stained specimens.

The best method of staining preparations of fresh blood is to dilute the drop with an equal quantity of ¾ per cent. salt solution, to which a little methyl violet has been added. This stains the nuclei of the leucocytes and the blood plates. It also brings out nucleated red corpuscles if these are present.

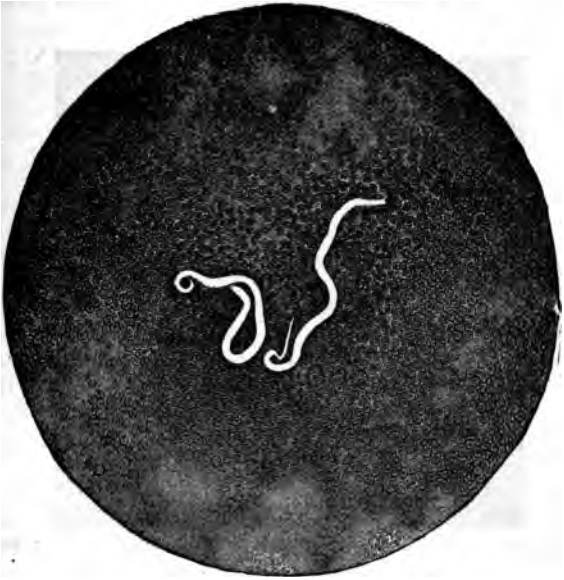

Fig. 68.—Filaria nocturna ; × 160. (*After Patrick Manson.*)

Sometimes particles of pigment can be noticed amongst the corpuscles. This is the condition known as **melanæmia.** It is found occasionally in chronic malaria.

The **spirillum** of relapsing fever can be recognised by this method, and also the **filariæ sanguinis hominis.** The latter can be seen, with a low power, moving about among the red cells.

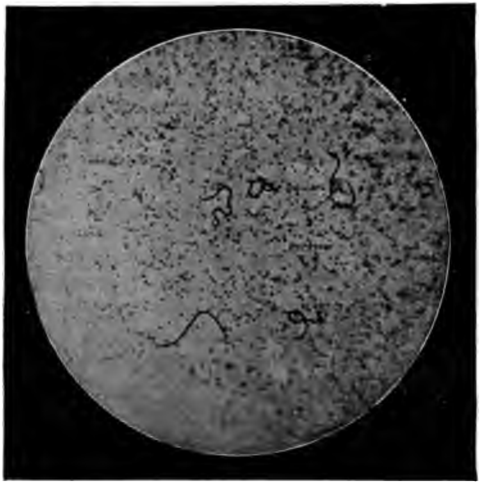

Fig. 69.—Embryos of filaria nocturna in blood; × 50. (*From an original microphotograph.*)

They average about $\frac{1}{75}$ in. in length, and are about as broad as a red blood corpuscle (Figs. 68, 69). They remain alive for a surprisingly long time even at ordinary temperatures, especially if the preparation is kept from drying by being sealed with a little vaseline. The following is the method recommended by Manson as best suited for their demonstration :—

Spread out a thick drop of blood on a slide by means of a needle, and allow it to dry. It may then be preserved indefinitely. When the parasites are to be demonstrated, immerse the slide in a solution of one drop of saturated alcoholic fuchsin in 1 oz. of water. Stain in this for one or two hours. If, on examining the film, it is found that the blood is very deeply stained, one must decolorise by means of dilute acetic acid (four drops of acetic acid to 1 oz. of water). The specimen may be examined either wet or dry, and with or without a cover. On searching it with a low power, the filariæ will be recognised by their being very deeply stained. The preparation is apt to fade after a few days.

A more rapid result is obtained by staining the film for half a minute in a 2 per cent. solution of methylene blue. It is then decolorised a little with dilute acetic acid as above described, and examined with a low power while wet. If a permanent preparation is desired, the film is allowed to dry and a drop of balsam and a cover glass applied.

2. **Blood cells fixed but unstained.**—This is the best method for studying changes of shape and size in the cells; 2 per cent. osmic acid is the most useful fixative. Place a drop of it on the finger or lobe of the ear, and prick the skin through it. The blood is fixed as soon as it exudes, and the mixed drop may be examined immediately. The addition of a little methyl green to the osmic acid helps to bring out the nuclei of the leucocytes. Permanent preparations may be obtained by running in a drop of glycerine under the cover glass.

This method shows very well the blood plates and the size and shape of the red corpuscles. Normally, of course, the latter are all disc-shaped and practically uniform in size. In some diseased conditions, however, this ceases to hold good. Thus there may be a number of red corpuscles present which are decidedly smaller than normal (microcytes), whilst others, again, may be considerably above the average size (megalocytes). Or, the red corpuscles may be distorted—spindle-shaped, indented, budded, etc. This is known as *poikilocytosis.* It is apt to occur in some forms of anæmia, especially in pernicious anæmia.

3. **Examination of blood in films.**—Films may be made either on slides or on cover glasses. The former have the advantage of being more easily manipulated, but cover glasses give the best results. The slides should be of colourless glass, thin, and with ground edges. The cover glasses should be ¾ of an inch square, and as thin and flexible as possible. It is important that both slides and cover glasses should be entirely free from grease. To ensure this they should be dropped one by one into an enamelled iron dish containing 10 per cent. chromic acid and boiled for 20 minutes. They should then be tipped into a shallow basin, and water allowed to run on them till the washings are colourless. After this they are covered with spirit and finally transferred with forceps to a wide-necked stoppered bottle containing absolute alcohol. When required for use they should be picked out with forceps, excess of alcohol drained off, and the remainder got rid of by passing through a flame. They should finally be rubbed with a clean handkerchief.

If ordinary slides or cover slips have to be cleaned in a hurry, glacial acetic acid, followed by water and alcohol, gives good results.

How to make films.—(1) *On cover slips.* The surface of the cover slips must on no account be touched by the fingers. They may be held by their corners between the thumb, middle, and index fingers, but it is preferable to use forceps—a clamp forceps for *the lower cover* slip and a fine-pointed forceps for the upper. Clean, dry, and prick the lobe of the ear; wipe away the first drop of blood, and when another about the size of a large pin's head has appeared touch its apex with the upper cover slip and lightly drop it on to the surface of the lower. Wait a second till the drop has had time to spread itself out, and then *slide* the two slips apart, avoiding any pressure or lifting. The lower slip will usually be found to have the better film on it.

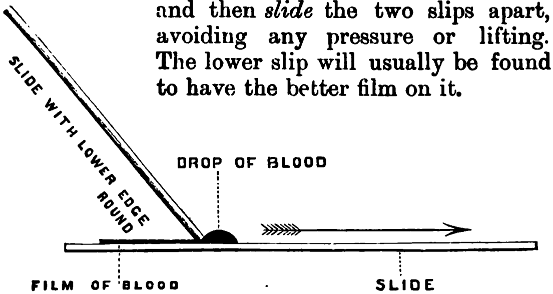

Fig. 70.—Diagram showing method of making a blood film by means o two slides.

(2) *On slides.* If one of the slides has a smooth, round edge it may be used to "plane out" the drop of blood upon the other, as shown in Fig. 70.

Better results are obtained by the use of gutta-percha tissue, or cigarette paper.

Take a piece of *smooth* gutta-percha tissue about 2 inches long and 1 inch broad. Fold it slightly along its long axis so that one side becomes somewhat convex. Pass one end of the convex surface lightly across the top of the drop of blood, and immediately lay it flat on the slide near one end. When the drop has spread itself out draw the gutta-percha tissue flatly along the surface of the slide. If cigarette paper * is used, cut strips across its long axis, each about half an inch broad, and as long as the paper is wide (Fig. 71). Pass the edge of the strip across the summit of the drop of blood, lay it on the slide, and, when the drop has spread, draw it along. Use a fresh strip for each slide.

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* The "Tarlene" or "Zig-zag" papers are best. Ordinary glazed notepaper will also do, but cigarette paper is preferable.

Fixation of the film.—Films may be fixed either while still wet or after drying. The former method has the advantage of fixing the leucocytes in their natural shape, and not flattened out as they are apt to be in dried films, and is also better for showing up details of nuclear structure; on the other hand, the dry method is rather more convenient.

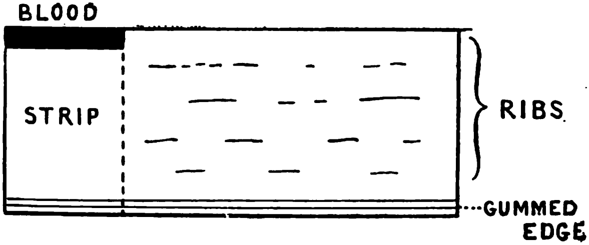

Fig. 71.—Cigarette paper prepared for making blood films. (*After Howard and Pakes*

(1) The best method of fixing films whilst still *wet* is that proposed by Gulland. He uses the following fluid :—

Saturated solution of eosin in absolute alcohol	25 cc.
Pure ether	25 cc.
Solution of corrosive sublimate in absolute alcohol (2 grms. in 10 cc.)	5 drops.

Some of the fluid is placed in a flat dish, and the cover slips are floated upon it wet side downwards; 5–10 cc. is sufficient for four cover glasses.

Films on slides may be plunged into a wide-necked bottle containing the fluid. Fixation is complete in three minutes, but it does no harm to leave the films for even 24 hours. Wash thoroughly in water, stain for a minute in saturated watery methylene blue, wash, and dehydrate with absolute alcohol, clear in xylol, and mount in xylol balsam.

Red corpuscles are stained pink, all nuclei deep blue, the blood-plates a fainter blue. The bodies of the leucocytes are in various shades of pink, the eosinophil and basophil granules being well brought out. Organisms are also well stained.

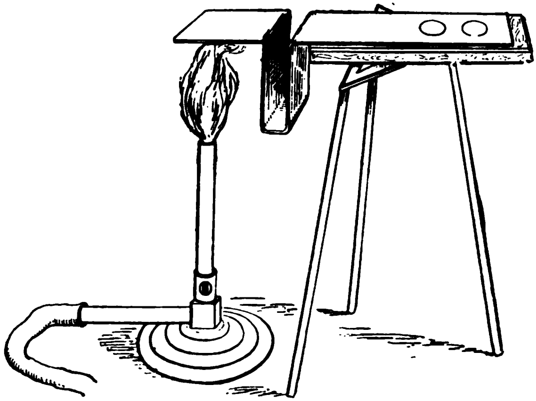

Fig. 72.—Copper plate for fixing blood films by heat

Pus, sputum, etc., may also be examined by the above method, but the fixation time for these should be longer.

(2) *Dry methods.*—Properly made films should dry in a few seconds after they are made. The

process may be hastened by waving them about in the air. They may then be fixed by one or other of the following methods :—

(*a*) *Heat.*—One may use a copper plate, one end of which is heated by a lamp or Bunsen. After about a quarter of an hour the temperature of the plate is uniform, and by dropping on water one can find the point at which it boils briskly, which will be 100–110° C. Lay the films face downwards at this point and leave them for 10 minutes. A convenient form of the apparatus is shown in Fig. 72.*

If a small copper oven is obtainable it is preferable to the above arrangement.†

For ordinary stains an exposure of two minutes to a temperature of 110° C. is sufficient, but if one desires to bring out the granules very clearly, as in using the triacid stain, an exposure of at least ten minutes is necessary. Preparations which have been overheated fail to stain well, whilst in those which have been insufficiently heated the staining is apt to be diffuse and, if triacid is used, in the tone of the acid fuchsin.

(*b*) *Chemical Fixatives.* Of these there are several. The most convenient are :—

Formol Vapour. An exposure of ten minutes is sufficient.

Absolute Alcohol and Ether. Equal parts, for two hours.

Formol and Alcohol. Various strengths have been proposed, but a mixture of 10 parts of formol to 90 of absolute alcohol will be found to answer very well (Gulland). The dry films are floated in this for three to five minutes, though a slightly longer exposure does no harm. Slides may be plunged into a wide-necked bottle containing the mixture. Wash in tap water. Stain (*see* below), wash again, dry and mount.

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* To be obtained from Messrs. Watson, High Holborn, W.C.

† The best form is a modified Victor Meyer's apparatus, with a roof of thin plate-copper and heated by toluol.

How to stain the film.—No single method of staining is sufficient for all purposes. For all-round work a combination of methylene blue and eosin is perhaps best, but logwood and eosin give a very good general impression of a film, whilst for finer work the triacid is the best mixture.

(*a*) *Logwood and eosin.* Films fixed by any of the above methods should be stained in Meyer's hæmalum or in Delafield's hæmatoxylin for about one minute, washed in *tap* water and counter-stained for a moment with saturated watery or a strong solution of alcoholic eosin, washed, dried, and mounted in balsam.

(*b*) *Methylene blue and eosin.* Any of the above methods of fixation may be employed, but formol vapour or formol-alcohol are the most convenient. It is well to remember that eosin discharges methylene blue much better than the last-named does the former. Stain in strong watery or alcoholic eosin till the film is bright pink. Wash, counter-stain with saturated watery methylene blue, controlling the depth of staining under the low power of the microscope.* (Half a minute is usually long enough.) Wash in water, dry and mount in xylol-balsam.

(*c*) *Triacid stain* (Appendix, 25). The best results are obtained in films fixed by heat, or formol-alcohol. Stain for from one to five minutes. (The exact time required varies in different films.) Wash in *distilled* water, dry, and mount.

Special methods. Of these the following may be briefly described :—

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* If over-stained with methylene blue, dip for a moment in 1 per cent. acetic acid and wash in water.

Jenner's method.[*] The stain[†] consists of a ½ per cent. solution of a specially prepared crystalline compound of methylene blue and eosin in pure methyl alcohol. Films are made as cover slips in the usual way. So soon as they are dry a few drops of the solution are poured on, and they are covered with watch glasses to prevent evaporation and precipitation of the stain. Pour off in one to three minutes, rinse in *distilled* water till pink. (This takes 5–10 seconds.) Dry rapidly high over a flame or by waving in the air. Mount in xylol-balsam. If tap water is used for washing, dip the film in it for a moment only, and dry between blotting paper or high over a flame.

In a successful film the red corpuscles are terra-cotta coloured; nuclei are blue, platelets mauve, the granules of polynuclear cells and myelocytes red, mast cells dark violet, bacteria, filarial and malaria parasites blue. If washed in *tap* water the red colour is more pronounced, the nuclei of the white corpuscles are pale, and the dark blue of the bodies of the lymphocytes stands out in contrast to the now pale nuclei.

This method is rapid and convenient, but in the authors' hands, at least, the results have not been quite constant, and the solution does not keep well unless very carefully corked.

Michaelis' method.[‡] In this method the neutral compound formed by the union of methylene blue and eosin is kept in solution by acetone. The following solutions are required:—

(1) A one per cent. watery solution of crystallised

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[*] *Lancet*, February 11th, 1899.

[†] Supplied by R. Kanthack, 18, Berners Street, W.; and by Messrs. Baird & Tatlock, Cross Street, Hatton Garden, E.C.

[‡] *Deut. Med. Woch.* (Vereinsbeilage, No. 27), 1899.

methylene blue (free from zinc chloride), 20 grms.; absolute alcohol, 20 grms. Mix.

(2) A one per cent. watery solution of eosin, 12 grms.; acetone, 28 grms. Mix.

The solutions are kept separately, and when required 1 cc. of each is mixed and the film floated on it face downwards. At first they become blue, but gradually this gives place to a reddish tint. This change must be watched for by taking out the film for a second from time to time and looking at it by transmitted light. The moment the pink tint has appeared, wash in distilled water, for if the process is allowed to go on the nuclei will become decolorised again. From fifteen seconds to five minutes at most will be the time required, but the exact period varies in different films.

Michaelis uses absolute alcohol (¼–24 hours) as his fixative, but good results are also obtained with films fixed by a short exposure to heat.*

This method is very rapid, and we have obtained some beautiful results with it, but the eosin solution requires to be freshly prepared at intervals of a few days, or it loses its power. Care also must be taken that the acetone is perfectly neutral. If these precautions are observed it is one of the best of all methods for clinical work.

Examination of the film.—In a good film the corpuscles should be spread out evenly, no rouleaux being seen. Even with the low power the white cells can be recognised by their stained nuclei, and some idea of their relative numbers gained. For the minute examination of the white cells a high power, and preferably an immersion lens, is requisite. In many cases it is important to make a "**differential**

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* Michaelis' method does not succeed well with films which have *been exposed to* formol.

count" in order to ascertain the relative numbers of the different varieties of leucocyte. For this purpose several hundred cells must be counted, which, with a little practice, can be done in a quarter of an hour.*

The following are the varieties of leucocytes found in normal blood (Plate IV., Fig. 1), with their relative proportions:—

1. Leucocytes with multipartite nucleus and very fine neutrophil granules (so-called "polynuclears"), 70 per cent.

2. Leucocytes with round or branched nucleus and coarse oxyphil granules ("eosinophils"), 2–4 per cent.

3. Small leucocytes with round nucleus and no granules (lymphocytes), 22–25 per cent.

4. Large leucocytes with oval nucleus and no granules ("large mono-nuclears"). These pass into the "transitional forms" with notched nuclei, more deeply staining, and perhaps a few neutrophil granules. Together these amount to 2–4 per cent.

5. Polynuclear cells with intense basophil granulation† ("mast cells"), 0·5 per cent.

The alterations which occur in the relative proportions of these in *leucocytosis* have already been mentioned (p. 207).

In the *lymphatic* form of leukæmia an enormous increase occurs in the number of the lymphocytes. (Plate IV., Fig. 3).

In the *myelogenous* form of the disease the neutrophils, eosinophils, and mast cells are all increased, and in addition bone marrow cells ("myelocytes") appear in the blood. These are of large size, with a

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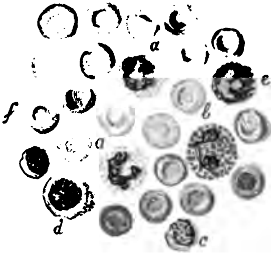
* To assist in making a differential count, Ehrlich recommends the use of an eye-piece with a square diaphragm to limit the field. This is made in two forms: (*a*) a simple diaphragm to drop into the micrometer eye-piece (Zeiss); (*b*) an ocular with a square diaphragm of iris pattern (Leitz). The latter is the more *convenient form.*

† *These are not stained* by the triacid mixture.

FIG. 1.—CELLS OF NORMAL BLOOD.



a. Finely-granular eosinophil leucocyte.
b. Coarsely-granular eosinophil leucocyte.
c. Lymphocyte.
d. Hyaline leucocyte, with fine amorphous points of basophil substance.
e. Finely-granular basophil leucocyte.
f. Red blood corpuscle.

FIG. 2.—CELLS FROM THE BLOOD IN MYELOGENOUS LEUKÆMIA.

a. Myelocyte; *a′*, with mixed granulation; *a″*, with double nuclei; *a‴*, myelocyte undergoing mitosis.
b. Normal coarsely-granular eosinophil leucocyte.
c. Normal finely-granular eosinophil leucocyte; *c′*, non-granular.
d. Atypical coarsely-granular eosinophil leucocyte.
e. Lymphocyte, with fine basophil amorphous points.
f. Normal finely-granular basophil leucocyte.
g. Atypical basophil myelocyte.
h. Nucleated red blood corpuscle; *h′*, undergoing mitosis.
i. Hyaline leucocyte, with fine basophil amorphous points.

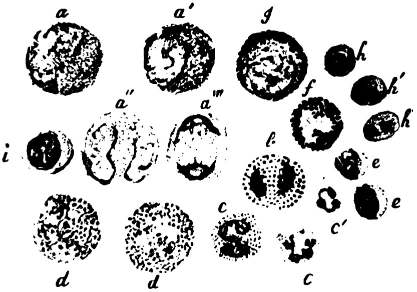
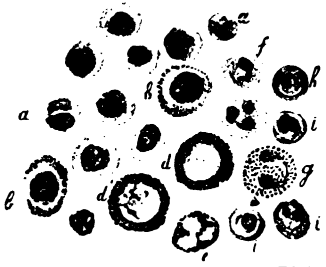


FIG. 3.—CELLS FROM THE BLOOD IN LYMPHATIC LEUKÆMIA.



a. Lymphocyte, with fine basophil amorphous points.
b. Hyaline leucocyte, with basophil amorphous points.
d. Atypical basophil leucocytes.
e. Normal finely-granular basophil leucocyte.
f. Normal finely-granular eosinophil leucocyte.
g. Normal coarsely-granular eosinophil leucocyte.
h. Normal red blood corpuscle.
i. Nucleated red blood corpuscl

PLATE IV.

(From an Original Drawing by Dr. Robert J. M. Buchanan.)

[To fac

single, faintly-staining nucleus, and contain granules which may be either neutrophilic or eosinophilic in reaction. (Plate IV., Fig. 2.)

Myelocytes are also found in the blood in *anæmia pseudoleukæmica infantum* and in some other conditions.

A relative diminution of the leucocytes is spoken of as *leukopenia*. It is found in enteric fever, splenic anæmia, and some other diseases.

The **red cells** may present various alterations in disease. The alterations may affect:—

(*a*) Their *size* and *shape*. Instead of the normal sized erythocytes small cells may appear, devoid of the usual central indentation (microcytes), or unusually large forms may be met with (megalocytes), particularly in pernicious anæmia. Instead of being rounded the corpuscles may become oval, pear-shaped, etc. These changes are spoken of collectively as *poikilocytosis*.

(*b*) The *staining power* of the cells may be altered. Thus instead of taking up eosin in a hæmatoxylin-eosin mixture, they may stain with the logwood too, and have a violet or even bluish tinge. This is spoken of as *polychromatophilia*. It is seen in various kinds of anæmia, and is believed to indicate a degeneration of the red cells.

(*c*) *Nucleated forms* may appear. If these are of the same size as ordinary red corpuscles they are spoken of as *normoblasts*. They can be distinguished from lymphocytes (for which at the first glance they are apt to be mistaken) (1) by the more homogeneous and intense staining of the nucleus, (2) by the presence round the nucleus of a cell body which stains red, (3) by their smoother contour.

Megaloblasts are very large nucleated red corpuscles. They may be even four times as large as an ordinary red cell. They have a relatively small faintly-staining nucleus, and a large cell-body, which often exhibits polychromatophilia. Megaloblasts are a characteristic feature of the blood in pernicious anæmia.

Microblasts—nucleated forms smaller than an ordinary red corpuscle—are found in traumatic anæmias, and are of no great significance.

Parasites in the blood.—We have already described the way to look for **filariæ** in the blood (p. 218). Of this parasite there are several species which are the embryos of corresponding parental forms. The embryos live free in the blood; the parental forms are found in the tissues and lymphatics. In one species the embryos are present in the blood during the night only (*F. nocturna*), in another only by day (*F. diurna*). In yet another form (*F. perstans*) they are always present. The blood in suspected cases should therefore be examined both during the day and during the night. For the diagnosis of the different species special works must be consulted, but the chief points to attend to are: (1) The time when the parasites are present in the blood; (2) the nature of their movements; (3) whether or not they possess a sheath; (4) the shape and character of their extremities.

To recognise the **parasite of malaria**, proceed as follows:—

Prepare some perfectly clean and very thin cover slips, and remove all traces of grease in the manner already described (p. 220). Cleanse the skin of the finger-tip or ear with soap and water and then with alcohol and ether. Make a *small prick* in the skin. Wipe away the first drop of blood, leaving a perfectly dry surface, so that subsequent drops will not run. Squeeze out a tiny drop about the size of a large *pin's head.* *Touch the apex of this drop with the centre of a cover glass*, and immediately drop it, face

Halitosis (bad breath cont.) Index.

IV Constitu. Diseases
1. Diabetes – fruity
2. Pyaemia. Pneu. abscess – fetid
3. End of Critical illness – cadaveric

V Nervous Disorders
1. Hysteria [illegible] Curry
2. Paresis ,,
3. Epileptics "pineapple or violets"
4. Menstruation (Mayer)

VI Kidney Diseases
1. Uraemia – urinous
2. Dis. of the Tubules (Bartel)

VII Blood Diseases
1. Scurvy – excessively foul
2. Pseudo Leukaemia – Mayer

IX Poisons
1. Opium – narcotic
2. Chloral [illegible]
3. Arsenic
4. Phosphorus garlicky
5. Bismuth
6. Hg [illegible]
7. Pb
8. Carbolic Acid
9. [illegible]
10. Iodides

X Drugs
1. [illegible]
2. [illegible]

XI Foods

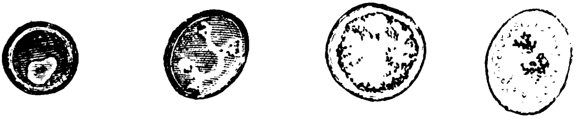


FIG. 1.

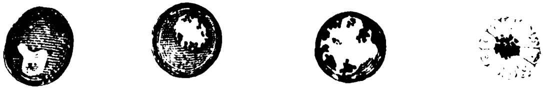


FIG. 2.

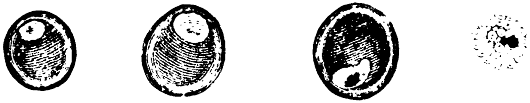
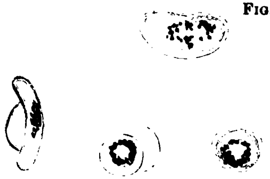


FIG. 3.



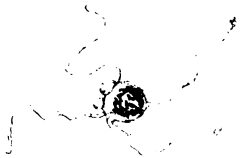


FIG. 4.

FIG. 5.

PLATE V.—THE BLOOD IN MALARIA. Showing different forms an stages of development of the parasite. (*After Thayer and Hewetson.*)

FIG. 1.—The parasite of tertian fever.
FIG. 2.—The parasite of quartan fever.
FIG. 3.—The parasite of æstivo-autumnal fever.
FIG. 4.—Crescentic and oval bodies.
FIG. 5.—Flagellated forms.

[To face p. 23]

downwards, on a perfectly clean slide. Make several such preparations, and reject all those in which rouleaux are present. It is absolutely essential that the red corpuscles should lie flat. Examine with a $\frac{1}{12}$ immersion lens and rather feeble illumination. Look in the red corpuscles for the presence of small black specks, often rod-like and showing slow movements of translation (Plate V., Figs. 1, 2). These are surrounded by clear areas. One may also see in the centre of some of the red cells clear amœboid areas which show no pigment (Plate V., Figs. 1, 2). Rosette forms may also be visible (Plate V., Fig. 2). These forms of the parasite are always present in cases of malaria which have not had quinine. Other varieties are only met with in some chronic cases. Of these there are two chief forms: (1) The cresentic, (2) the flagellated (Plate V., Figs. 4 and 5). These are easily recognised. The cresentic bodies are highly refractile, rather longer than a red blood corpuscle and about 2μ in diameter. Particles of pigment may be recognised in the parasite and also in some of the ordinary leucocytes.

The examination of blood for the malarial parasite demands some care.* Manson says that one must devote half an hour to the examination of a slide before pronouncing on the absence of parasites in it. The quartan form of the parasite (Plate V., Fig. 2) is distinguished from the tertian (1) by being smaller in size, (2) by its pigment granules being darker, (3) by its showing fewer segmenting forms.

If it be desired to stain the parasite, one should use for the purpose a concentrated solution of methylene blue in 0·6 per cent. salt solution. A tiny drop of this is placed on the skin, and the puncture made through it. The mixed drop is then examined as

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* Parasites are most abundant in the blood about 8 to 12 hours *after the rigor.*

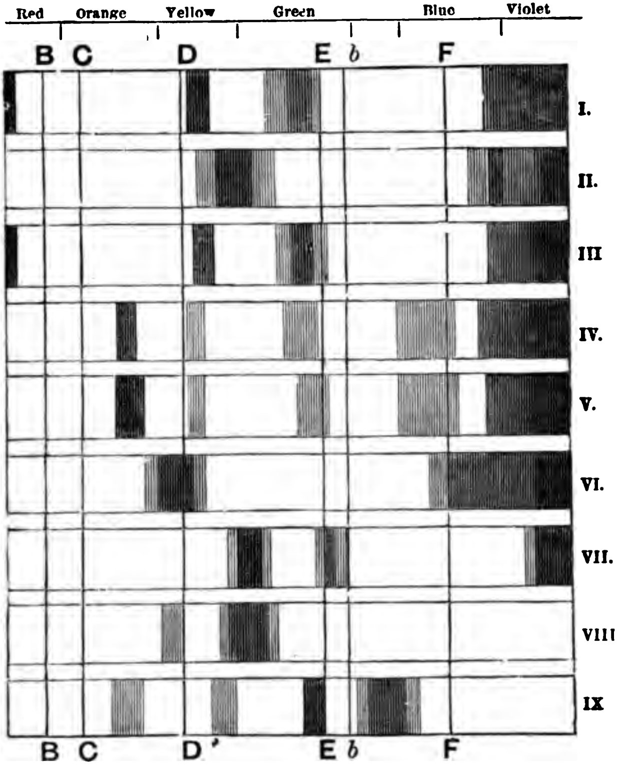

Fig. 78.—Spectra of hæmoglobin and its derivatives.

I., oxyhæmoglobin ; II., reduced hæmoglobin ; III., carbonic oxide hæmoglobin ; IV., methæmoglobin (in acid solution) ; V., acid-hæmatin ; VI., alkali hæmatin ; VII., hæmochromogen ; VIII., hæmatoporphyrin (in acid solution) ; IX., hæmatoporphyrin (in alkaline solution).

above. The parasite stains blue, while the red cells remain uncoloured.*

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* Patrick Manson has described a special method by which *the flagellated form of the* parasite can be successfully stained (*Brit. Med. Journal, 1897,* vol. ii., p. 68).

To get *permanent preparations*, proceed as follows:—Stain a fixed film for two seconds with very dilute eosin (0·5 cc. concentrated alcoholic eosin to 500 cc. water), wash, pour on some Löffler's solution (Appendix, 22), diluted one in four of water. Wash this off after a few seconds, allow the film to dry, and mount in balsam.

The parasite is stained blue and the red cells pink.

The examination of the blood for **bacteria** is considered in Chapter XIV.

Carbonic oxide in the blood.—In cases of suspected carbonic oxide poisoning, the presence of the gas may be detected by spectroscopic examination. Some blood is obtained by the application of a cupping-glass, and is freely diluted with distilled water. The solution has a cherry-red colour. Place some of it in a thin, flat glass tube, and examine with a hand spectroscope. Direct the instrument, as in all such examinations, towards a white cloud, and not towards the sun. Two bands (Fig. 73) are seen (bands of carbonic oxide hæmoglobin) occupying very much the position of the oxyhæmoglobin bands. They are distinguished from the latter by the fact that addition of a few drops of sulphide of ammonium produces no alteration in them.

Various methods have been proposed for the clinical estimation of the **specific gravity of the blood.** The simplest is the following:—

A mixture is made of chloroform and benzol in such proportions that the specific gravity of the fluid, as taken with a sensitive urinometer, is 1,060. Some of this is placed in a tall glass vessel, and a drop of blood added to it from a hæmocytometer pipette. If the drop remains suspended without either rising to the surface or sinking to the bottom, the specific gravity of the blood is the same as that of the mixture. If the drop floats, add benzol out of a burette, stirring well with a glass rod, until the drop remains suspended. If it sinks, add chloroform till a similar result is attained. Then take the specific gravity of the mixture with a urinometer and the result will give the specific gravity of the blood.

The normal specific gravity of blood is about 1,060. Variations are not of much clinical value. The specific gravity is always in proportion to the amount of hæmoglobin—a low specific gravity means little hæmoglobin. The ratio is so constant that one can tell the percentage of hæmoglobin by taking the specific gravity.

The clinical estimation of the **alkalinity of the blood** can only be accomplished by the use of rather elaborate methods, the results yielded by which are not of sufficient importance to justify their description in a work such as this.*

The **coagulability of the blood** can be estimated with a fair degree of accuracy by means of Wright's coagulometer.† The instrument consists of a series of fine tubes of equal calibre which are kept at a uniform temperature. Blood is drawn into each of the tubes at definite intervals, and, after the lapse of varying periods of time, one blows down the tubes in succession. If the blood can no longer be blown out, coagulation has occurred. The interval between the filling of the tube and the occurrence of coagulation is known as the "*coagulation time.*" At a temperature of 18·5° C. the coagulation time of a healthy individual is about four minutes.

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* For a discussion of the methods employed in the clinical estimation of the alkalinity of the blood and the results which they have yielded, *see* a paper by one of the authors (Dr. Hutchison) in the *Lancet*, March 7th, 1896.

† The instrument is supplied, along with full directions for use, by Hawksley, 357, Oxford Street, London, W. Another method of estimating the coagulability of the blood has been proposed by Brodie & Russell (*Journal of Physiology*, xxi., 1897, p. 403). The authors have had no experience of its use.

CHAPTER VI.

RESPIRATORY SYSTEM.

SECTION I.—ANATOMY.

(Plates I., II., III.)

THE following anatomical facts must be borne in mind when the lungs are examined:—

1. **Borders of the lungs.**

Right lung.—The anterior border passes forwards, downwards, and towards the middle line from the apex, which, situated at the level of the neck of the first rib, corresponds posteriorly with the seventh cervical spine. Behind the sternum, at the level of the second rib, it has nearly reached the middle line, and passes directly downwards to the level of the junction of the sixth costal cartilage with the sternum, where it turns rather abruptly to the right to pass outwards as the lower border. The lower border meets the right parasternal line at the level of the upper border of the sixth rib, the mammary line also at the level of the sixth rib, the axillary lines at the seventh and eighth ribs, the scapular line at the tenth rib, and at the side of the vertebral column reaches as far as the tenth interspace or eleventh rib.

Left lung.—From the apex to the level of the fourth costal cartilage the anterior border passes in a direction which corresponds with that of the right lung. At this point it bends rather suddenly outwards, thereby leaving part of the anterior surface of the heart exposed, and passes in an arched line outwards and downwards to reach the sixth rib a *little external* to the parasternal line. From this point the lower border passes backwards along a line corresponding to, but a little lower than, that of the lower border of the right lung. The lower borders of both lungs are convex towards the abdomen. In forced respiration they may vary in level to the extent of two or even three inches, according to the phase of the respiratory cycle. In quiet respiration the difference between the extremes is only about 1 cm.

2. **Lobes of the lungs.**

It is often important to know the limits of the individual lobes of the lungs. This may be done by drawing a line from the second dorsal spine to the sixth rib in the mammary line; this corresponds to the upper border of the lower lobe. A second line, drawn forwards on the right side from the centre of this line to meet the sternum at the level of the fourth costal cartilage, will mark the boundary between the upper and middle lobes.

Obviously, therefore, the greater part of each lung, as seen from behind, is composed of the lower lobe, only the apex belonging to the upper lobe; whilst the middle and upper lobes on the right side, and the upper lobe on the left, occupy most of the area in front. In the axillary regions, parts of all the lobes are accessible.

The **bifurcation of the trachea** corresponds in front with the lower part of the manubrium sterni; behind with the disc between the fourth and fifth dorsal vertebræ.

The **reflected pleural sacs** reach decidedly lower than the inferior borders of the lungs, whose limits they overstep for about two inches in the mammary, nearly four inches in the mid-axillary, and one and a-half inches in the scapular lines. The sinus thus formed lies on the left side above the resonant stomach *cavity, and therefore*, should it become distended with *fluid, as in* cases of hydrothorax, a dull area will be discovered at a part where the healthy percussion note is tympanitic. The anterior reflection of the left pleura below the fourth rib is considerably nearer the middle line than the anterior border of the left lung; hence in emphysema, when the lung presses forward into this available space, the area of absolute cardiac dulness is greatly encroached upon.

With reference to the correspondence of points in front and at the back, Quain gives the following relations as existing during expiration:—

"The upper margin of the sternum is on a level with the disc between the second and third dorsal vertebræ; the junction of the manubrium and body is opposite the fifth dorsal vertebra; and the xiphisternal articulation generally corresponds to the lower part of the ninth dorsal vertebra."

The **scapula** is a useful landmark posteriorly. Its upper angle, when the arms hang by the side, is generally on a level with the disc between the first and second dorsal vertebræ, the root of the spine with that between the third and fourth dorsal vertebræ, and its lower angle with the body of the eighth dorsal vertebra.

In reference to the ribs, the upper angle of the scapula just covers the second rib; the lower angle reaches as low as the seventh interspace or eighth rib.

The twelfth rib cannot always be felt. It is not safe, therefore, to count the ribs from below upwards.

For convenience in description, the thorax is mapped out into regions, as follows:—

(*a*) **Three central regions anteriorly.**

Suprasternal, from the cricoid to the upper border of the manubrium.

Superior sternal, from the upper border of the manubrium to the level of the third chondrosternal articulation.

Inferior sternal, from third chondrosternal articulation to lower end of sternum.

These three regions are bounded laterally by the lateral sternal lines and their upward continuations.*

(*b*) **Five antero-lateral regions** on each side.

Supraclavicular, bounded above by an oblique line from the side of the cricoid to the outer end of the clavicle, below by the clavicle.

Clavicular, composed of the area occupied by the clavicle.

Infraclavicular, bounded above by the clavicle, below by a horizontal line at the level of the third chondrosternal articulation.

Mammary, from the lower edge of the infraclavicular area to the level of the sixth chondrosternal junction.

Inframammary, below that level.

These regions extend outwards to the anterior axillary line.

(*c*) **Two lateral areas on either side.**

Axillary *Infra-axillary*	meeting each other at the level of the sixth rib.

(*d*) **Four regions at the back on either side of the spine.**

Suprascapular.

Scapular, subdivided into supra- and infraspinous.

Infrascapular, and

Interscapular.—The position of the dorsal regions is sufficiently defined by their names.

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* *Sometimes* the sternal regions are classified as "episternal" *and "xiphisternal."*

SECTION II.—INSPECTION.

Inspection determines :—

- (A) Form of chest.
  - (*a*) Healthy.
  - (*b*) Symmetrical chests with features indicating proclivity to disease.
    - The alar chest.
    - The flat chest.
  - (*c*) Symmetrical chests with features indicating past disease.
    - The rachitic chest.
    - The pigeon breast.
    - Harrison's sulcus.
  - (*d*) Symmetrical chests with features indicating present disease.
    - The barrel-shaped chest.
    - Bilateral retraction.
  - (*e*) Unilateral changes.
    - Enlargement.
    - Diminution.
  - (*f*) Local changes.
    - Bulging.
    - Retraction.
    - Funnel-shaped depression.
- (B) Movements of chest.
  - (*a*) Respiratory.
    - (1) Rate.
    - (2) Rhythm.
    - (3) Type.
    - Character (*see* also Chapter II.).
      - Amount of expansion.
      - Unilateral fixation.
      - Local lagging.
      - Local indrawing and bulging.
  - (*b*) Non-respiratory. Pulsations (Chapter IV.).

(A) The **shape of the thorax** depends partly on the curvature and obliquity of the ribs, partly on the curves of the spinal column. The curvature of the sternum results from the relations of these factors in each case.

When the ribs are normally curved, the more horizontally they lie, the more nearly does a cross section of the chest approach the form of a circle, the wider are the intercostal spaces, and the more obtuse does the subcostal angle become ; whilst, on the contrary, *increasing* obliquity of the ribs leads to narrowing of the intercostal spaces, to increasing ellipticity of the cross section of the chest, the major axis lying transversely and the minor axis in an antero-posterior direction, and at the same time the subcostal angle becomes more acute. In a healthy male the angle is about 70°, in a female about 75°. The variations may amount to 10° above or below these averages. When there is lateral curvature of the spine, the chest is rendered asymmetrical; when the spine is unduly concave forward, other changes are produced, which will be dealt with subsequently.

The **ideal healthy chest** will conform to the following description: It is bilaterally symmetrical, its contours are smooth, it has no deep hollows, and at most shows only a slight recession below the clavicles. In cross section it is an ellipse, broader from side to side than from front to back in the proportion of about 7 to 5; and its general shape is ellipsoidal, with the longest axis vertical. In children the cross section is much more nearly circular.

The sternum, which is convex from above downwards when viewed from the front, lies at the bottom of a shallow groove known as the sternal furrow, formed by the pectoral muscles of each side. The junction of the manubrium with the body of the sternum exhibits a slight angular projection (angulus Ludovici), sometimes visible, almost always palpable. The sternal furrow ends below, at the level of the seventh costal cartilage, in the infrasternal depression (or scrobiculus cordis). A slight hollow below the clavicle marks the separation between the divisions of the pectoralis major; it should not be deep, and ought only to be distinct when the muscle is made to contract. A second hollow, which is much more distinct, separates the pectoralis from the deltoid. This fossa lies farther from the middle line, and is known as the infra-clavicular (or Morenheim's) fossa. It becomes very marked in many cases of phthisis.

The shape in the mammary regions depends greatly on the degree of development of the mammary gland and on the amount of subcutaneous fat. In the adult male the nipple is usually situated about 4 in. from the middle line, in the fourth intercostal space.

In actual practice it is very rare to find a chest which is perfectly symmetrical. Generally the right side is rather more capacious than the left, and the spinal column almost always has a slight degree of lateral curvature. In inspection of the chest the examiner must never be content with viewing it from one aspect only. He should first look at it from the front, then from the side, thereafter from the back, and, finally, he should look over the shoulders from behind and above, so as to see the profile of a horizontal section of the thorax. The last method is very useful in detecting lack of symmetry or unequal expansion on the two sides. The neck, especially as regards the manner in which it is set on the chest, and the epigastrium should be inspected at the same time as the thorax.

Deviation from the normal form may either affect the whole of the thorax or localised parts of it. The **abnormal shapes** of the chest as a whole may be grouped in three classes, according as they indicate merely a proclivity to lung disease, a history of former disease, or the existence of present disease. The first class contains the alar and flat chests; the second the rickety chest, the pigeon breast, and the chest with Harrison's sulcus; the third includes the barrel-shaped chest and the hollow or retracted chest.

In these groups the changes affect both sides of *the thorax, and* so the symmetry remains undisturbed.

In other instances the morbid conditions at work may lead to unilateral changes in the shape of the chest, one side having its volume either increased or diminished, and being otherwise deformed. Lastly, the chest may exhibit local deviations from the normal form, due generally to local disease

I. **Symmetrical chests with features indicating proclivity to lung diseases ("Phthinoid" Chests).**—The two forms which belong to this class are the alar and the flat chest.

1. The **alar chest** is one where the vertebral borders of the scapulæ project unduly, and the shoulders droop. The cause of this appearance is to be found in the obliquity of the ribs, which makes the projection of their curves and angles in the horizontal plane more sharp, leads to a long and rather shallow thorax, and is associated with a long neck and prominent throat.

2. The **flat chest** is due to a loss of the forward convexity of the costal cartilages, which become more or less straight. As a result, the sternum is less distant from the vertebral column than usual. The flat chest is often, but not always, associated with the alar form.

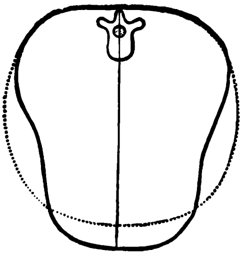

Fig. 74.—Cross section of rachitic chest. (*Gee.*) The dotted line represents the normal outline for the same age.

II. **Symmetrical chests with features indicating past diseases** (and not seldom predisposing to pulmonary disease).

This group contains a number of forms, but only a few need be considered here.

1. The **rachitic chest.**—In rachitis the bones *are less rigid* than usual, and so are more readily

deformed by any applied force. From the nature of the disease the part that yields most readily is where the bone and cartilage meet, and therefore, when any cause prevents the free access of air to the lungs during inspiration, this part bends inwards before the pressure of the external air. A vertical groove is thus formed in this region, and persists even after the cause which first led to its production has disappeared. The section of a rachitic chest is shown in the accompanying figure, where the depressions situated at a little distance from either side of the sternum are easily recognised. When the rachitic condition is severe the line of least resistance becomes so weak that no unusual obstruction to inspiration is necessary in order to produce the grooves; the slightly lower air pressure within the thorax, which is necessarily present during inspiration, being sufficient to lead to its formation (Fig. 74).

2. The **pigeon breast.**—Here, in consequence of some obstruction (often quite trivial) to inspiration at a time of life when the ribs are soft, they become straightened in front of their angles; where, owing to their smaller degree of curvature, they are most readily deformed by external pressure. The result is that the sternum becomes unduly prominent and projects beyond the plane of the front of the abdomen, so that there is a sharp angle at its lower end. At the same time the cross section of the chest ceases to be elliptical, and approaches a triangular form, the angles being

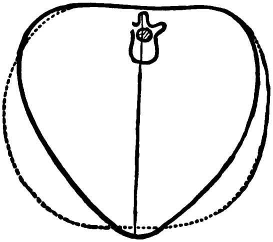

Fig. 75.—Cross section of pigeon breast. (*Gee.*) The dotted line represents the normal outline.

situated at the sternum in front, and at the costal angles behind (Fig. 75).

3. **Harrison's sulcus.**—This is a transverse constriction which, beginning at the level of the xiphisternum, passes outwards and slightly downwards. It seldom reaches as far as the mid-axillary line. This deformity is due to the same cause as the last, but either the obstruction has been slighter, or the bones have been more fully hardened.

The depression is therefore limited to the most yielding part of the chest, and this corresponds to the region where the cavity is widest. Lower down than the sulcus, the liver and other abdominal viscera had supported the chest wall and so prevented it from being drawn inwards; whilst, higher up, the greater curvature of the ribs had enabled them to withstand the external pressure.

The three deformities just described are frequently found together in one individual, and cases of pigeon breast almost invariably exhibit a well-marked Harrison's sulcus.

III. Symmetrical chests with features indicating present disease.

1. **In emphysema** the increased volume of the lungs demands increased space for their accommodation. To provide this, the ribs are less obliquely set than usual, whilst the spine becomes unduly concave forwards, and the sternum is much more arched than under normal conditions, whilst the angle of Louis becomes extremely prominent.

Since this increase of volume is accomplished by the mechanism which is provided for the expansion of the chest in inspiration, the latter can only be effected by movements of the chest as a whole, and so the accessory muscles of respiration have to take the *place of the intercostals*, thereby becoming abnormally *conspicuous*, whilst the diaphragm has a considerable excess of work imposed upon it. The chest of emphysema is described as "**barrel-shaped.**"

Kyphosis may produce a form of chest which closely simulates that of emphysema.

2. **Bilateral hollowing** is an extreme case of the flat chest already described, and is caused by the existence of phthisis.

IV. **Unilateral changes in the shape of the chest which affect the whole of one side.**—These may result either in unusual bulging or in retraction.

Unilateral enlargement is due either to the presence of fluid * or gas in the pleura, or to increase in volume of one lung due to a tumour, or compensatory hypertrophy.

Diminution of volume results from shrinkage of the lung. This may be caused by phthisis, or it may be the result of adhesions formed during an attack of pleurisy with effusion. Collapse of a lung from obstruction of the bronchi may produce a similar result.

Before connecting these changes with disease of the lungs or pleura, the observer must ascertain that no **scoliosis** exists; for the rotation of the vertebræ in this condition leads to a deformity which, when inspected only from the front, it is almost impossible to discriminate from those just described.† Abdominal disease must also be excluded.

V. **Local changes,** affecting only part of either side.

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* The presence of fluid does not always cause enlargement of the affected side of the chest, although it often does so. Sometimes indeed that side is smaller. The explanation is not very obvious, but it must be recollected that the fluid is primarily accommodated by displacement of the lung, and only later, after the elasticity of the lung has been exhausted, will the contour of the thorax be altered.

† Scoliosis may, however, be induced by the retraction of a lung in a *young subject.*

1. **Bulging.**—In emphysema the apices may produce an unusual fulness above the clavicles, and in pleural effusion, especially when of a purulent nature, the interspaces which lie in the area of effusion often bulge considerably; these phenomena may accompany a general enlargement, or occur without it.

Tumours of various kinds, and disease of the heart, may be the cause of localised bulging.

2. **Shrinking.**—In phthisis one or both apices are often contracted, and thereby a hollowing is produced above the clavicles. A similar condition occurs also in the infra-clavicular regions, which may exhibit marked hollowing. This is particularly noticeable in Morenheim's fossa.

To detect either bulging or flattening (as well as diminished expansion) it is important to look tangentially along the chest either from above the shoulders or upwards from below. Both in phthisis and other wasting diseases the interspaces are very sunken, and the ribs prominent, in consequence of the malnutrition of the muscles and subcutaneous tissues.

Not unfrequently a local shrinking is due to adhesions of the pleura resulting from a former attack of pleurisy.

3. A **funnel-shaped depression** is sometimes found in the lower part of the middle line of the thorax on front. Sometimes it is congenital, or it may be developed in infancy with or without any obstruction to respiration being present. It extends in some cases as high as to the third rib. A similar depression—though seldom of such magnitude—is found as a trade deformity in shoemakers.

(B) **Movements of the thorax.**

The movements of the chest during respiration also demand attention, and the rate of movement, its *rhythm, its type,* and its amount, must be noted.

1. The **rate** for an adult in health is about eighteen or twenty respirations per minute, but there is a wide margin on either side of these figures. Increased rapidity may result from exertion, nervous excitement, fever, defective aëration of the blood, whether this be due primarily to cardiac, pulmonary, bronchial, and laryngeal causes, or to some alteration in the oxygen-carrying power of the blood. It may also arise from the association of pain with all attempts at respiration, as in pleurisy and peritonitis, when the breathing becomes shallow, and must therefore be more frequent to make up for the slighter expansion.

The **ratio between respiration and the pulse** is important. In health it is about 1 to 4; in pneumonia respiration may occur almost as frequently as the pulse; in certain cases of narcotic poisoning the ratio may become 1 to 6 or 7.

2. The **rhythm** varies very considerably even in health, and if the act be performed consciously it may become very irregular. Hence it is important to study it when the patient is off his guard, as only then can accurate observations be made. Either inspiration or expiration may be unduly prolonged; the former being commonly associated with laryngeal or tracheal, the latter with bronchial and pulmonary diseases. A peculiar type, where successive respirations gradually get deeper and deeper till a maximum is attained, and then fall off again until a pause of complete apnœa occurs, to be followed by another wave of gradually deepening and then diminishing respiration, is known as **Cheyne-Stokes breathing.** The pause may last for fully half a minute, though it is often shorter, and the whole cycle is usually completed in less than two minutes. It is very conspicuous when the patient who exhibits it is *asleep, or is unconscious*; but is apt to be overlooked

if the patient is awake, and particularly if he is talking. Apart from completely typical Cheyne-Stokes respiration, various modifications, more or less nearly approaching it, occur.

There is another form of respiration which is often mistaken for Cheyne-Stokes breathing, but which is really different. In this form, instead of a gradual increase in the depth of respiration from the apnœic pause to the middle of the cycle, the deep breathing begins suddenly, and gradually diminishes, until the apnœic pause is reached, thereafter to recommence once more with full vigour.

3. **Type.**—Breathing may be more evidently performed by the upper part of the thorax; this is known as the **thoracic type of respiration.** It is found to a certain degree in women, but in its full development is associated either with paralysis of the diaphragm, or else as a result of its fixation from inflammatory causes or increased abdominal pressure.

In men and young children the **diaphragm** and **abdominal muscles** play the most important part in respiration; and in cases where the intercostal muscles are paralysed, or where some inflammatory and painful condition, such as pleurisy, or pleurodynia, exists in the thorax, the breathing may be wholly abdominal in type.

In health the male type of respiration may be described as **abdomino-thoracic**, the female as **thoracico-abdominal,** or almost purely thoracic.

The **presence of pain** or **dyspnœa** should always be inquired for, and its exact nature noted. [*See* Chapter II.]

4. Regarding "**movement**" during respiration, the points to be noted are its amount, whether it is expansive in character, and whether it is similar on *the two sides* and over corresponding areas.

Amount of movement and expansion are by no means interchangeable terms; in emphysema the chest may move considerably, but there is little expansion.

In comparing the two sides it will often be found that deficient or absent movement betokens pleurisy with effusion, or non-expansion of the lung from consolidation or rigidity of its structure.

Local deficiency in expansion is frequently a very important indication of phthisis, or it may be due to lobar pneumonia, the former especially at the apices, the latter at the apex or base according to the situation of the disease.

Sometimes one part of the chest wall lags behind the rest during inspiration. Any such lagging is important as suggestive of disease. The existence of any **indrawing** of the chest wall or of the interspaces during inspiration, or of any **bulging** during expiration, must be noted. Both may occur physiologically, in which case the conditions are present over the whole chest, and are not very conspicuous, or they may result from pathological conditions, when they sometimes affect the whole thorax, at other times one side, and yet at others only appear locally. Examples of inspiratory indrawing are found in obstruction of the larynx (general), or in blocking of some of the smaller bronchi (local). One of the best instances of localised expiratory bulging is seen at the apices of the lungs in advanced emphysema.

SECTION III.—PALPATION.

Palpation determines :—

A. Form of chest [confirms or modifies the results of inspection *q.v.*].
B. Movements of chest.
    a, Respiratory [*vide* also inspection].
    b, Pulsations [Chapter IV.].

C. Vibrations
  - Palpable pleuritic friction.
  - Palpable râles.
  - Vocal fremitus.
    - increased.
    - diminished.
    - absent.

D. Tenderness.
E. Fluctuation.
F. Resistance of chest wall to compression.

Inspection of the thorax should go hand in hand with palpation and mensuration. In this way the observations made by the eye are confirmed and extended, in some respects greater accuracy is attained, and various facts which the eye cannot discover are elicited.

Palpation takes note firstly of the form and movements of the thorax; secondly, of vibrations or tremors which are communicated to the hand; and thirdly, of the behaviour under pressure of any pain of which the patient complains. Under the first head inspection is supplemented; under the second one learns something of the accompaniments—*e.g.* friction, or rhonchi—which interrupt the smoothness of the respiratory movement, and also of vocal fremitus, which serves to indicate the condition of the conducting media. The third enables one to detect the cause of many thoracic pains.

Before making a systematic examination, it is well to lay the hand on any part of the chest which presents an obvious swelling, or where the patient complains of pain. In doing so the observer should remember to look at the patient's face rather than at the part under examination, as he thus most quickly learns whether he is causing any avoidable suffering. Pain may be due to inflammatory conditions in the chest wall, to intercostal neuralgia, where, as a rule, specially painful spots can be discovered corresponding to the points where the branches of the affected nerves emerge through the fascia; to intercostal myalgia, where the pain is aggravated by pinching the affected muscle, or to pleurisy. In the case of the latter, pressure may considerably increase the pain by bringing the opposed surfaces of the inflamed pleura more firmly into contact. At the same time the nature of any swelling should be investigated. The hand will also supplement the information derived from inspection with regard to prominence of the intercostal spaces, and may occasionally detect fluctuation in them when there is pleuritic effusion. Fluctuation also occurs, and is much more distinct, when an abscess has formed in the chest wall. Such an abscess may be due to disease of the bones or soft parts forming the parietes of the thorax, or to pus which has broken through from the pleural cavity (*empyema necessitatis*). In the latter case the pus may often be driven back by gentle pressure, to reappear when the patient coughs.

When these preliminary observations have been completed, the observer should direct his attention to the **form of the thorax.** Here the hand is best aided by mechanical appliances, such as the cyrtometer, and by simple measurements. Tracings and measurements should be taken at the periods of full expiration and inspiration. In a well-formed adult male the girth of the chest at the level of the nipples should be 34 inches at the end of expiration, and should measure at least two inches more when a deep inspiration has been taken. Height, age, and build of course greatly modify these measurements, and insurance returns indicate that different races vary very considerably in chest girth. It is generally far more important to ascertain the increase of girth between expiration and inspiration, both full and ordinary, than to determine the exact circumference of the chest at either phase.

If the shape of a cross section of the chest is required, a tolerably efficient cyrtometer can be improvised by connecting two pieces of flattened composition gas-pipe, each about two feet long, by a hinge of elastic tube. The hinge should be placed over the spine, and the metal pipe moulded to the surface of the chest. It can then be opened at the hinge and closed again over a piece of paper, to which the outline should be transferred if a permanent record is desired. To prevent any risk of subsequent confusion, the back and right side of the tracing should at once be marked as such, and a line should be drawn from the position of the hinge to the point in front which corresponds with the middle of the sternum. The length of this line may be checked by a pair of callipers, as a precaution against accidental bending of the cyrtometer during its transference from the patient to the sheet of paper. For convenience in preservation a reduced copy of the tracing may be made by means of a pantograph; if this is done a note of the exact proportion between the original and the copy should be recorded on the latter.* There is no difficulty in applying the same simple instrument so as to obtain the outline of the chest in other planes than the horizontal. Thus, by placing the hinge above the shoulder, the two pieces of pipe may be carried down the parasternal line in front and in a corresponding line behind, whilst, by sharply bending their lower ends outwards, they may be made to cross each other in the axillary line and the point of intersection marked. The instument is then opened at the hinge and readjusted over the paper so as to yield the desired tracing.

The **nature of the respiratory movements** must next be studied. It is important to make certain that the two sides of the chest move to approximately the same extent. This is done by fixing the finger-tips of either hand at the patient's sides, and making the radial borders of the thumbs meet in the middle line in front of the chest. The hands being kept rigid, the patient is directed to take a full inspiration, when the distance of departure of the thumbs from the middle line indicates the extent of expansion of either half of the chest.

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* Various more elaborate instruments have been devised, but, though ingenious, they are cumbrous, and consequently not of great clinical use. The best of them is Dr Graham *Brown's perigraph.*

Sometimes one half of the thorax lags behind the other; this is readily detected by the hands no longer moving synchronously.

The movements at the apices may be similarly observed. In this case the physician stands behind the patient, and fixing his thumbs on the vertebræ, lets his fingers lie over the right and left lung apices reaching towards the clavicles whilst the patient breathes deeply. Thereafter one hand should be placed on front of the chest, and the other on the epigastrium. In health, as the chest expands, the epigastrium is also raised to a greater or less degree. If the epigastrium fall in with each expansion of the chest, there is reason to suspect paralysis or flaccidity of the diaphragm. Fixation of the diaphragm with immobility of the epigastrium during respiration is generally due to abdominal disease (*see* p. 57).

Vibrations may be detected by palpation. *For this purpose the palm of the hand should be applied flat on the chest*, and since the sensitiveness of the two hands is often unequal, *the same one should be employed on both sides.* In addition to the vibrations already referred to in the chapter on the heart, fremitus may be due to pleural friction, to catarrhal changes in the mucosa of the bronchi, leading to local *constrictions*, or to fluid in the bronchi or in pulmonary cavities. After the presence or absence of these forms of fremitus has been determined, the observer should study the **vocal fremitus,** or vibrations which the voice communicates to the chest wall. These are conducted from the larynx by the trachea and bronchi to the smaller tubes within the lungs, and thence through the lung tissue to the surface. Anything which affects the conducting power of the air-passages or lung tissue, or the interposition of additional materials through which the vibration must pass to reach the palpating hand, will obviously affect the intensity of the fremitus. To test the vocal fremitus, the patient is told to repeat "one, one, one," or "ninety-nine," in a clear voice. The hand placed on the thorax detects distinct vibration whilst this is done, and it must be determined whether the vibrations in corresponding areas on the two sides of the chest are approximately equal in intensity—not, however, forgetting that where the heart encroaches on the left lung the fremitus is necessarily much diminished—and also whether they correspond to what former experience has led the observer to recognise as normal for the region under examination, for a similar chest and like pitch and loudness of voice. **Vocal fremitus is increased** when the voice is of a deep pitch, when the chest wall is rigid, and often when it is thin, as also when the lung is consolidated, or contains a cavity near its surface. Since the right bronchus is wider and shorter than the left, whilst the septum separating the two bronchi occupies a position to the left of the centre of the trachea, the laryngeal sounds pass more freely along the right than they do along the left bronchus, and therefore the vocal fremitus is normally somewhat greater over the right lung than over the left.

Vocal fremitus is diminished when the pitch of the voice is high, when the chest wall is thick, and especially when there is much thickening of the pleura. It is greatly diminished, or totally absent, when the lung is separated from the chest wall by pleuritic effusion. The cause in this case is not that fluid is a bad conductor of sound or of vibration—the reverse is the case—but because the relaxed lung itself fails to convey the local fremitus, and so the vibrations never reach the fluid. In young persons and in female subjects the vocal resonance is different both in character and intensity from that which occurs in male adults. The differences are due to the different conformation and degree of rigidity of the thorax, and to the distinctive pitch and quality of the voice in each instance. The resistance of the chest to compression is best estimated by placing the hand over the sternum whilst the patient is lying down, and attempting to press it backwards towards the vertebral column. The rigidity naturally increases with advancing age, but observations also indicate that in certain diseases (*e.g.* in phthisis and in emphysema) the rigidity often becomes greater than usual. Where this is so the prognosis is rendered less favourable, as free expansion of the lung is hindered.

SECTION IV.—PERCUSSION.

Percussion determines :—

A. The boundaries of the lungs [topographical percussion].
B. The resonance of the lungs.
  (*a*) Normal variations in different parts.
  (*b*) Abnormal alterations.
    1. Quantitative { *Increase* [hyper-resonance]. *Diminution*, in varying degrees, from slight impairment to absolute dulness.

2. Qualitative :—
   - Tympanitic
     - high-pitched.
     - medium-pitched.
     - low-pitched.
   - Skodaic.
   - Boxy.
   - Cracked-Pot.
   - Bell sound (coin percussion).
   - Amphoric.

But little need be added here to what has been already stated in a previous chapter regarding the theory of percussion. It must be recollected that it is a most difficult task to give even a partial explanation of the phenomena observed from the standpoint of physics, and in practice it is rarely necessary to appeal to theory, as a long experience has enabled physicians to attach certain meanings, more or less empirically, to various percussion phenomena.

It may, however, help the student to appreciate the various sounds when he hears them, if a few of the main factors in their causation are recapitulated.

Firstly we have to consider the materials which produce the sound. These are the pleximeter, the chest wall beneath it, and the subjacent viscus so far as it comes within the range of action of the percussion stroke. The pleximeter sound, by the choice of a suitable material, may either be rendered insignificant, or, in consequence of its special qualities, immaterial in its effect on the resonance. The chest wall yields a sound varying with the part struck, and depending for its quality on whether sternum, clavicles, ribs, or soft parts underlie the pleximeter. The sound due to the wall is, however, quite subordinate to that of the organ lying beneath when the latter contains air, and when the percussion stroke is firm enough.

The **character of the sound produced** varies quantitatively and qualitatively, the *quantitative variations* depending on the force of the blow delivered, and on the capacity of the part struck to resound to the blow.

The **quality of the sound** depends on the particular vibrations which are elicited, and on the selective reinforcement of some of them by the resonance of the organs involved.

When the air in a cavity of sufficient size and appropriate shape is set into vibrations which are not modified by excessive tension of the containing walls of the space, the sound heard has a tympanitic character, but when the cavity is subdivided into a number of small loculi by numerous septa, more or less tense, a characteristic resonance, no longer tympanitic, is produced. Such conditions prevail in the healthy lung, and the observer must learn by assiduous practice to recognise its distinctive quality. In general terms this pulmonary resonance may be said to be low in pitch and clear in character.

In percussion over the lung we endeavour to ascertain three sets of facts: firstly, the position of the apices and lower border of the lungs, and also of that portion of the anterior border of the left lung which lies over the heart; secondly, the state of the lungs in regard to the quantity of air contained in their various parts, and the tension of their elastic framework; and, thirdly, whether they are unusually remote from the surface of the chest, the separation being due to thickened parietes, or fluid or gas in the pleural cavity.

The apices and borders.

Owing to the slope of the surface of the neck, resonance can usually be observed in health for one and a half to two inches above the level of the clavicle. The **apices** are either equally high above the clavicles, or the right may reach a shade higher than the left; if the right is a little lower than the left, or *the left* decidedly lower than the right, there is a probability of past or present disease in the lung whose apex fails to attain the normal limits. Should both apices be very low in level, there may be disease of both lungs. In emphysema both apices are generally found considerably higher than in health. When the examination is made the patient must look straight before him, not turning the head to the side away from the examiner, as this alters the tension of the muscles over the lung. The percussion stroke should be moderately strong, and care should be taken that it is delivered quite perpendicularly to the surface. Wherever there is any doubt of the apices of the lungs being normal the whole course of their upper borders should be determined. Commencing posteriorly at the level of the spine of the vertebra prominens, the limit of lung resonance in health passes outwards along a line which curves gradually upwards to reach the anterior border of the trapezius, about an inch and a half above the level of the clavicle. Thence it passes obliquely downwards and forwards until it approaches the outer border of the sternomastoid, when it inclines more directly downwards towards the clavicle. Sometimes it hardly reaches so far forward as the sternomastoid, at other times the line runs along the surface of the muscle. In cases where the tracheal resonance interferes with the precise delimitation of the lung, the difficulty may be avoided by making the patient open his mouth, thus altering the pitch of the tracheal percussion note.

The **lower border of the right lung** lies over the liver, and is thin; therefore its exact situation is best made out by light percussion. Posteriorly, however, the muffling due to the thick muscles and fat of the back makes it necessary to percuss more firmly. When the patient is obese, very heavy percussion with several fingers may be necessary in order to penetrate the parietes, and bring the lung tissue within the sphere of influence of the blow. In quiet respiration the lower border is found to lie in the mammary line at the sixth rib, in the mid-axillary line at the eight rib, in the scapular line at the tenth rib, and, nearer the vertebral column, as low as the tenth space.

On the left side the lower border overlaps the stomach, and so the transition is not from lung resonance to dulness, but to tympanitic stomach resonance. Posteriorly, however, the splenic dulness and the dulness of the various solid structures which lie below the lung near the spine are interposed, so that the conditions resemble those found on the right.

The position of the lower border corresponds pretty closely with that on the right side; it may, however, be found a trifle farther down. In old people the lower borders of both lungs extend beyond these limits, in children they do not reach them by about a rib's distance lower and higher respectively.

The **anterior border of the left lung** emerges from behind the sternum at the level of the fourth costal cartilage, and forms the upper and left limits of the area of superficial cardiac dulness.

The limits described are exceeded in very deep inspiration, and in diseases such as emphysema, where the volume of the air-containing lung is increased. In pneumothorax the lower border of resonance is often considerably below the limits assigned, and the character of the sound is different (p. 264).

The limits are not attained when the lungs are shrunken or consolidated, when increased abdominal pressure interferes with the normal level of the diaphragm, or when there is effusion in the cavity of the pleura. In this case, should the effusion be left-sided, instead of passing in the anterior axillary line *from lung* resonance to tympanitic stomach resonance, a band of dulness will be found between the two resonant areas; and since the lower limit of the pleural sac reaches nearly four inches* lower at this point than the inferior border of the lung, the dulness will pass downwards to a lower level than the normal lung resonance does, and Traube's area (p. 67) will be encroached upon (Fig. 76). In consolidation of the lung, on the contrary, this area will not be diminished.

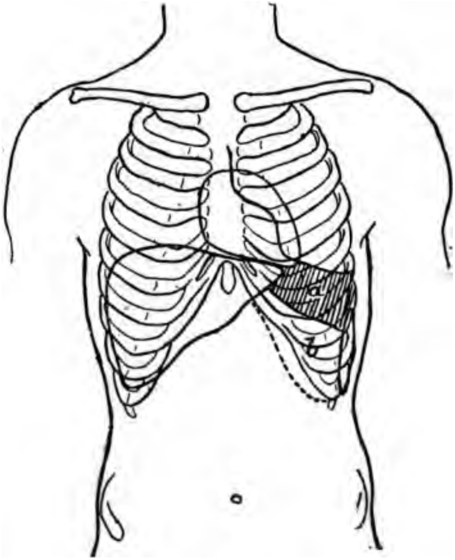

Fig. 76.—Traube's space in pleuritic effusion.
a, portion rendered dull; b, portion remaining resonant.

Having outlined the lungs, the character of the percussion sound over their various parts must be studied. Beginning in front, the examiner should tap lightly on the most prominent point of each clavicle—being careful to ascertain that the points examined correspond exactly with each other—and should observe the quality of the sound, and particularly determine whether under like conditions of percussion the effects on either side are identical. Thereafter the other corresponding areas on either side should be carefully compared, many points being systematically percussed in each area. The presence of the heart will obviously interfere, in certain parts of the left side, with the development of a sound resembling that on the corresponding point on the right.

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* *In the* cadaveric condition Luschka gives it as 10 cm.

When the front has been fully examined, the observer should percuss in both axillary and infra-axillary regions—the patient meanwhile holding his hands joined above his head; lastly, the various areas posteriorly should be worked out; the patient, if able to sit up, being instructed to fold his arms and bend slightly forwards.

It is most essential at all parts of this examination that the patient's attitude be a comfortable one, so as to avoid any unilateral strain on his muscles, and that his arms and shoulders should be placed symmetrically. The head must not be inclined to either side.

If any of the regions are unusually hollowed so that the finger cannot be readily adapted to them, a small cork will be found to make a good pillar pleximeter.

Should the patient's chest be unsymmetrical (*e.g.* from spinal curvature), the observer must not expect equal resonance on the two sides.

In a healthy individual the resonance in the various regions will exhibit the following characteristics :—

Apices.—Clear, not very intense, as the vibrating mass is small, and tending to have a slight tympanitic quality added as the trachea is approached. The right apex is usually rather less resonant than the left.

Clavicular regions.—*Sternal end.* Clear, moderately intense, with tympanitic element due to trachea. *Centre.* Clear, more intense than in supraclavicular or outer clavicular regions. Devoid of tympanicity. *Outer end,* as centre, but less intense.

Infra-clavicular regions.—Clear and intense. Slightly tympanitic near sternum.

Mammary regions.—Here there is naturally a difference between the two sides; on the right, the lung is encroached on in the lower part of this area by the liver; on the left, the heart occupies a good deal of the space, and the stomach note is elicited through the thin lung at the lower part. In general, however, the pulmonary resonance is clear and fairly intense, except where the neighbouring organs come within the range of vibration. The chest wall here is thicker from the presence both of the pectoral muscles and the mammary gland, and the sounds elicited are consequently more muffled.

In the **infra-mammary regions** the sounds are greatly influenced by the neighbourhood of the liver, the colon, and the stomach. The lung-sound, however, is clear, though not intense, the thin layer of lung becoming rapidly emptier of resonance as its lower border is approached.

In the **axillary regions** the sound is more intense and clear than elsewhere, diminishing however in intensity at the lower part of each lateral area.

Posteriorly, the great masses of muscle which clothe the back muffle the resonance and make it feebler; and therefore firmer percussion, often with *several fingers* is required. The scapular region is most muffled, the infra-scapular least so. The interscapular and supra-scapular regions are intermediate in quality.

In disease, the resonance may be affected (1) quantitatively and (2) qualitatively.

Resonance is increased in emphysema (slightly), but at the same time the pitch is raised by the greater tension of the chest wall, and this in some cases not only prevents the increased resonance from being observed, but almost suggests dulness.

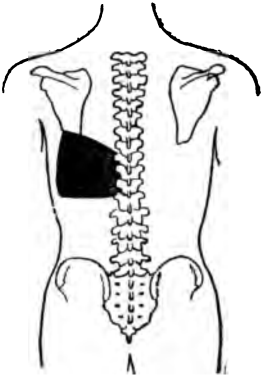

Fig. 77.—Pleurisy, with effusion seen from behind. (Case 1.)

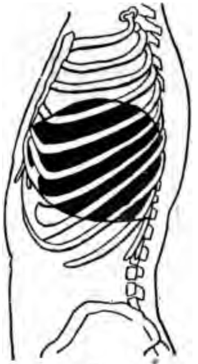

Fig. 78.—Pleurisy, with effusion, seen from the side. (Case 2.)

When the lung tissue is relaxed, but still contains air, the effect of the septa which subdivide the air columns is for the most part abolished, and the sound becomes distinctly tympanitic. At the same time the resonance is increased in intensity. This is sometimes called **Skodaic resonance**, and occurs above the level of a pleural effusion, or in the upper portion of a lung whose lower lobe is affected by pneumonic consolidation. When air has found its way into the pleural cavity, the sound is as a rule intensely tympanitic, unless the air be under considerable pressure. A characteristic form of high-pitched tympanitic resonance may be heard, in pneumothorax, by percussion over the front of the chest with a couple of coins; one being used as a plessor and the other as a pleximeter, whilst the observer listens at the back of the patient. In very marked cases the sound is soft and musical, and has been compared to the chiming of a distant church bell; in cases that are less pronounced it approximates rather to the stroke of a hammer on an anvil when heard a long way off.

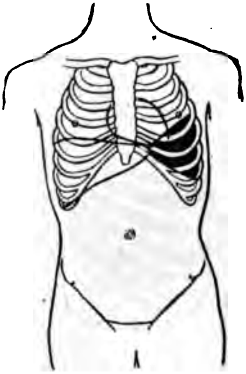

Fig. 79.—Pleurisy, with effusion, seen from the front. (Case 1.)

Cavities in the lung, or the presence of large or medium bronchi within range of the percussion stroke, likewise cause the sound to become tympanitic. A tympanitic sound, which may closely resemble that caused by the presence of a vomica, is heard when the portion of lung which lies between the trachea or primary bronchi and the surface becomes consolidated. This sound is sometimes called "Williams's tracheal resonance," and is most frequently discovered in the first or second intercostal spaces near the sternum. It is by no means an uncommon phenomenon.

Resonance is diminished in cases where the pleura is thickened, or where there is consolidation of the lung, either of a whole lobe, as occurs in pneumonia, or of small patches, as in early phthisis. In the latter instance a particular strength of percussion stroke will in each case be found to develop the dulness to the best advantage, according to the size of the solid patch and its distance from the surface. When fluid is present, as in hydrothorax or pleurisy with effusion, the *dulness is absolute*, and an unusual sense of resistance is experienced by the pleximeter finger. In pleurisy with effusion the upper limit of the fluid generally follows a curved line, as is shown in the accompanying figures (Figs. 77, 78, 79).

In the case of patches of solid lung substance, the airless portion is often surrounded by a shell of tissue in which the septa are relaxed. The result of percussion over this composite arrangement is to produce a sound whose resonance is less intense and emptier than the healthy lung would yield, whilst what is left of it assumes a sub-tympanitic quality. The effect is described as a **boxy or wooden sound.**

Several peculiar sounds which are produced by percussion in pathological conditions remain to be noted.

Cracked-pot sound.—This is due to a sudden expulsion of air through a constricted orifice. It occurs in cases where percussion is practised over a cavity which communicates with a bronchus of moderate size, and is most distinct when the mouth is opened. It has a hissing character, combined with a chinking sound like that produced by shaking coins together. It is also heard in certain cases of thoracic fistula, and occasionally in pneumothorax, as well as in the relaxed lung above the level of fluid in pleurisy, and near the consolidated area in pneumonia. If healthy children are percussed whilst they are crying, a cracked-pot sound is often produced.

Amphoric resonance.—This phenomenon is due to the selective reinforcement of certain vibrations by a large cavity; by this means the overtones are *accentuated* and die out more slowly.

The following alterations in percussion sounds may be observed under certain pathological conditions; their explanation is for the most part simple from a physical standpoint:—

(*a*) Tympanitic resonance, when due to the presence of a pulmonary cavity which communicates with a bronchus, is raised in pitch when the patient opens his mouth.

(*b*) The pitch of the percussion sound over a cavity varies with the position of the patient. The most obvious explanation is that, if the cavity be partly filled with fluid, this varies in position, and so alters the shape of the cavity when the patient changes his attitude. Other factors, however, often enter into the interpretation of this change.

(*c*) The resonance over a cavity becomes higher in pitch during inspiration, and lower during expiration. The phenomenon depends on the tension of the wall of the cavity.

(*d*) In pneumothorax the metallic resonance is higher in pitch when the patient is lying down than when he sits up.

In certain conditions of malnutrition the muscles on the front of the thorax are unduly irritable. In these circumstances a light tap over the sternum produces fibrillary contractions, at some distance off, in the pectoral muscles. This phenomenon often occurs in phthisis, and is known as myotatic irritability.

SECTION V.—AUSCULTATION.

Auscultation determines:—

A. Character of respiratory sounds.

- (*a*) Vesicular breathing (rustling in character)
  - Normal.
  - Puerile.
  - Harsh.
  - Jerky or cog-wheel.
  - Feeble or absent.
  - With prolonged expiratory murmur.
- (*b*) Bronchial breathing (guttural [ch] or aspirate [ha] in character)
  - I. ordinary
    - Low-pitched (cavernous).
    - Medium-pitched.
    - High-pitched (tubular).
  - II. Amphoric (with an echoing quality added)
    - Low-pitched.
    - Medium-pitched.
    - High-pitched.
- (*c*) Indeterminate or broncho-vesicular breathing.

B. Vocal resonance.

- (*a*) Quantitative changes
  - *Increase*
    - Slight.
    - Marked, Bronchophony.
    - Extreme, Pectoriloquy.
  - *Decrease*
    - Slight.
    - Marked.
    - Entire absence
- (*b*) Qualitative changes
  - Ægophony.
  - Amphoric resonance.

C. Accompaniments.

- (*a*) Râles
  - Dry. (Rhonchi)
    - Sibilant or high-pitched.
    - Medium-pitched.
    - Sonorous or low-pitched.
  - Moist (Crepitations)
    - *Non-resonant* or toneless
      - Fine.
      - Medium.
      - Coarse.
    - *Resonant* (metallic) or consonant
      - Medium.
      - Coarse.
- (*b*) Friction sounds.—Fine, medium, coarse.
- (*c*) Splashing sounds.—(Hippocratic succussion).

In auscultation three observations must be made at each point examined: First, the character of the breath sounds; second, the character of the vocal resonance; and third, the presence or absence of other sounds.

In order to make these observations with facility, the examiner should attend to the attitude of the patient, which must be as symmetrical and as unconstrained as circumstances will permit. This is easily attained when the patient can sit up; but if he is unable to do this he should be rolled round first to one side and then to the other, in order that the back, and especially the bases of the lungs, may be thoroughly examined. The student must remember that in serious cases great injury may be done to a patient by too prolonged an examination. Care must be taken, especially when an ordinary single stethoscope is used, that the chest-piece is accurately applied, and that no undue pressure is exerted. The patient must be directed to breathe through the nose, regularly and fairly deeply, but not noisily.

There are two typical varieties of breath-sound, both of which are audible in health at certain parts of the chest, and these must be carefully studied. The first is known as vesicular breathing, the second as bronchial. The former is heard over healthy lung tissue, the latter over the trachea and main bronchi.

In **vesicular breathing,** which can be heard typically in the axillary and infra-scapular regions of a healthy individual, the following facts will be noted :—

The inspiratory sound is fairly intense, and is audible during the whole of the act. The pitch is low, and the quality is characteristic, being somewhat rustling. It is this quality which is specially described as vesicular.

The expiratory sound follows that of inspiration without a distinct pause—unless, as not unfrequently happens, the patient holds his breath for a second at the end of inspiration—it is less intense than the inspiratory sound, is lower in pitch, and lacks the vesicular quality, being more of a simple blowing sound. It only remains audible during the earlier part of the expiratory phase, and under normal conditions the inspiratory sound is heard for at least twice as long as the expiratory.

To learn to recognise **bronchial breathing,** the student should listen over the trachea, though he must not expect to hear so intense a type of bronchial respiration when he subsequently examines a diseased lung.

The inspiration sound is moderately intense. It becomes inaudible shortly before the end of inspiration. Its pitch is much higher than that of vesicular breathing, and the quality is blowing or hollow, with *a guttural or aspirate* intonation.

The expiration sound is generally more intense than the inspiratory; the pitch is often higher; the duration extends through the greater part of expiration, being as long as, or even longer than, the inspiratory sound, from which it is divided by the silent period that marks the end of inspiration. In quality it exactly resembles the inspiratory sound, being aspirate or guttural in character. This quality is sometimes described as "tubular," but the same name is also applied to one of the *varieties* of bronchial breathing, and so is better avoided.

The principal variations which can be detected in **vesicular breathing** are as follows :—

1. **Puerile.**—The sounds are harsher than in the adult, but have a similar duration.

2. **Harsh**, with prolongation of expiration, the character, however, remaining vesicular. This frequently indicates loss of elasticity of lung tissue; hence it often occurs in early phthisis, but may occur in bronchitis.

3. **Jerky, interrupted, or "cog-wheel" inspiration.**—Here the sound is not continuous, but occurs in waves or sharp jerks. This indicates irregular expansion of the alveoli, due to unequal elasticity in various parts of the lobules, and is therefore not unfrequently present in early phthisis. It may also result, however, simply from nervousness, and to carry any weight as a physical sign it must be well marked even on deep inspiration. Even then, however, it may mean little or nothing, and should only take a very secondary place.

4. **The respiratory murmur may be feeble,** or even inaudible. In quiet breathing the expiratory sound is often quite absent. By making the patient breathe more deeply the murmur may be rendered audible. When marked, this condition *may indicate* defective expansion.

Total disappearance of the breath sounds usually occurs below the level of fluid in pleuritic exudation, because the relaxed lung does not conduct sounds well, and hence they are not conveyed to the fluid, which is itself a comparatively good conductor (*see* p. 281). If, however, there be only a small quantity of fluid present, the sounds may be faintly heard as the relaxation of the lung tissue is less pronounced. Occasionally it happens that when a considerable quantity of fluid has accumulated, the breath sounds, instead of disappearing, become loud, and possess a marked bronchial character. In such cases the vocal resonance is also loud, but is usually more or less ægophonic. This exceptional state of matters is most commonly observed posteriorly over the lower lobe of the lung, and may be due to collapse of part of the lung enabling the stronger vibrations which are present in a bronchus to be transmitted to the fluid with less loss of intensity than if they had first required to pass through air-containing lung.

With regard to *prolongation* of the expiratory sound, it must be recollected that in certain diseases, such as emphysema and asthma, the expiratory act is performed much more slowly than in health. In consequence of this, the respiratory sound may also be prolonged; hence in these diseases prolongation of expiration conveys a meaning different from the usual one.

Some patients are habitually shallow breathers, whilst others naturally breathe deeply. The ear can detect these variations, partly by the duration of the respiratory sounds, and partly by their intensity. The depth of breathing as estimated by auscultation is sometimes known as the "**respiratory excursion.**"

Bronchial breathing may be subdivided into three varieties, according as the laryngeal respiratory sound is conveyed to the ear through consolidated lung from the larger, medium, or smaller air-passages, each of which, by reinforcing certain elements of that sound, gives it a distinctive character.

In the first case we have **low-pitched bronchial breathing,** the more capacious tubes responding best to the deeper-toned elements of the laryngeal murmur; in the second case the pitch is **medium**; in the last it is **high.** Low-pitched bronchial breathing is heard pathologically over moderately large cavities in the lungs, and is hence sometimes called *cavernous;* high-pitched bronchial breathing is heard when consolidation has occurred round the smaller tubes, as in pneumonia, where the most perfect examples of bronchial breathing may often be found. Here the character is aspirate rather than guttural. This variety is often known as *tubular breathing.*

A special variety of bronchial breathing exists under diseased conditions, and is known as **amphoric respiration.** It resembles the sound produced by blowing across the mouth of a bottle or the muzzle of a gun. The sound, when analysed, is found to consist of one or more low-pitched fundamental tones and a number of high-pitched overtones. It is characteristic of a direct communication between a bronchus and either a considerable cavity with fairly smooth walls or a pneumothorax. The latter condition yields the best examples.

In cases where the resonance of a bronchus is within earshot of the observer, but where at the same time air-containing lung intervenes between the bronchus and the chest wall, the sound of the breathing combines both vesicular and bronchial elements, one or other type predominating according to the exact relations in each case. This variety of breath sound is known as **broncho-vesicular or indeterminate.** In such cases it is usually the expiratory sound which has more or less of a bronchial character. It occurs in health in certain regions where anatomical causes favour its production, especially near the roots of the lungs behind, and in the upper portions near the middle line in front. The resonance of bronchi which lie so deeply in the chest as to be completely muffled by the thick layer of lung tissue that separates them from the ear may become audible when the tissue around them becomes solidified, and thus conducts the sounds more effectively. If the consolidation reach to the surface of the lung the breathing will be bronchial, but if it does not extend so far, bronchial breathing will be heard through the vesicular breathing which is still being produced between it and the surface.

The breath sounds must be ausculted in the various regions that have already been examined by percussion, their character in each noted, and similar regions on the two sides of the chest compared; care being taken that the points examined correspond accurately to one another.

The second series of observations is directed to **the intensity and character of the vocal resonance.** It varies in intensity even in health on the two sides and over different areas of the lung, being louder on the right side and more intense the nearer the stethoscope is to the larger bronchi. When the patient repeats the words "one, one, one," or "ninety-nine," the ear receives from the chest no distinct impression of the syllables pronounced, but only a buzzing sound, whose intensity depends on the loudness and depth of the patient's voice and on the conductivity of his lungs. Other words or sentences may be used instead of the above, but, on the whole, these are well adapted to produce satisfactory and uniform vibration of the chest, and are therefore suitable for the purpose of comparing different points with one another.

An easy way of keeping a standard of intensity in the mind when examining is to conceive of the sound taking rise at different distances from the observing ear.* In some cases the sound is very distant. This is equivalent to "marked decrease' in vocal resonance. Sometimes the sound appears to be produced at a little distance from the chest-piece of the stethoscope. In this case the resonance is slightly decreased, and, to make certain of this, a comparison should at once be made with the corresponding point over the other lung. In fact, as in percussion and palpation, so in estimating resonance—each point examined on one side of the chest should be at once compared with the corresponding point on the other side. Vocal resonance of normal intensity generally conveys the impression of being produced just at the chest-piece of a single stethoscope. If it seems to be nearer the ear than this, the resonance is increased. When it is near the ear-piece of the stethoscope the increase is "marked," and the condition is often described as **bronchophony.**

If the words become articulate and seem to be spoken right into the auscultator's ear, it will generally be found that even whispered words are clearly heard. This condition is called **pectoriloquy.** Increased resonance occurs when, through any cause, the lung substance conducts the sound waves set up by the voice more clearly than usual from the bronchi. Consolidation is the commonest cause of increased lung conductivity. Bronchophony occurs when a moderately large bronchus is surrounded by a layer of solid lung reaching to the chest wall. Pectoriloquy is fairly characteristic of a cavity of some size communicating with a bronchus. Cases do, however, occur where a certain degree of pectoriloquy is heard over the front of the upper lobe of the lung when the lower lobe is compressed, as, for instance, by pleuritic effusion. Care must be taken that the articulate sounds do not reach the observer either through the other ear or by the patient's lips being directed towards the stem or ear-piece of the stethoscope.

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* The same method is to some extent applicable to the examination of breath sounds. It is rather arbitrary, as it does not take account of the differences which are normally found in different areas of the chest; still it is serviceable, especially for beginners.

For reasons already explained, vocal resonance is either entirely abolished or much diminished where a layer of fluid separates the lung from the chest wall (*see* p. 270). It is also diminished in cases of thickened pleura, and of emphysema.

Under certain conditions the *quality* of the vocal resonance undergoes modification. Pectoriloquy has already offered us an example of such a modification, but a noticeable change also occurs in pneumothorax, when an **amphoric** or metallic **echoing resonance** is imparted to the voice, as well as to the breath and heart sounds. Another alteration in the quality of the vocal resonance is observed in some cases of pleurisy. When the quantity of effusion is rather scanty, so that the lung is only separated from the chest wall by a thin layer of fluid, a nasal or bleating character may be imparted to the voice. This bleating tone is observed much more frequently at the back, near the lower angle of the scapula, or between that point and the axillary line, than it is over other regions of the thorax. It is known as **ægophony,** and is probably due to collapse of the bronchial tubes.*

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* Dr. Stone (quoted in Fagge's "Principles and Practice of Medicine," 3rd ed., vol. i., p. 940) considers that the peculiar quality of the voice is due to the fundamental tone being

The last series of observations is directed to the detection and recognition of various **adventitious sounds.**

These may arise either in the lung or in the pleura, and it must never be forgotten that sounds by no means very dissimilar may be produced by the friction of the stethoscope on a hairy chest wall; but the latter can usually be suppressed by moistening the skin. The accompaniments arising in the lung and bronchi themselves first demand attention.

Such accompaniments are collectively known as **râles,** and are subdivided into dry râles and moist râles. **Dry sounds,** known also as **rhonchi,** are produced in the air-passages, and are due to partial obstruction of their lumen either by swelling of the mucosa or by the presence of tough secretion. The mechanism of their production is thus comparable with that to which cardiac murmurs owe their existence.

They vary in pitch, the variations being in a great measure due to the size of the tubes where they take origin. The smaller tubes are the seat of high-pitched or **sibilant rhonchi,** and these are most abundant during the later part of inspiration; the medium-sized tubes yield medium-pitched rhonchi, and the larger bronchi produce the deep-toned or **sonorous rhonchi,** which are heard early in inspiration, and may be almost continuous. Dry sounds are characteristic of bronchitis, but are also found quite apart from any definite bronchitis in certain other diseases of the respiratory system, such as cases of phthisis when the bronchial tubes get plugged.

Moist râles, also called **crepitations,** are discontinuous sounds, and are produced either in the alveoli or in the bronchioles and bronchi. They produce on the ear a noise like the bursting of

intercepted by the effusion to a much greater degree than the overtones, which thereby become unduly conspicuous.

smaller or larger air-bubbles, and indicate the presence of fluid secretions in the air cells or tubes. They are classified as fine, medium, and coarse or bubbling.*

Fine crepitations are caused by the opening up of collapsed alveoli whose walls have been agglutinated by the exudation of a little fluid secretion. This at first causes them to adhere, but, as the air pressure gradually increases during the movement of inspiration, the adhesion at last gives way suddenly, and allows air to enter. The separation of the walls is accompanied by a cracking sound, which can be imitated by separating the moistened forefinger and thumb near the ear. When this condition occurs in a number of alveoli, the combined effect is to produce a sound of fine crepitation. It occurs only † near the end of inspiration, as is to be anticipated from its mode of production, and indicates the presence of exudation in the alveoli of the affected part of the lung. Fine crepitations are very characteristically present during the first stage of pneumonia, and in acute congestion from any cause; they also occur in early miliary tuberculosis. After atelectasis they are occasionally heard, and in œdema of the lung they occur in association with bubbling râles which are caused by the simultaneous presence of fluid in the bronchi.

Medium crepitations occur chiefly in the smaller bronchi, and are audible at the end of inspiration and the beginning of expiration. They are caused by the air bubbling through fluid secretion which has been poured out into the lumen of the bronchi.

Coarse bubbling crepitations occur in the larger divisions of the bronchi, and may be heard at almost any phase of respiration; they may be quite continuous in their occurrence. Coarse crepitations may also originate in pulmonary cavities.

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* The term "crepitation" is sometimes restricted to the first variety, the others being called fine and coarse bubbling râles.

† Rarely a few sounds closely resembling fine crepitations are *heard during expiration.*

Sometimes the râles are **non-resonant or toneless.** In this case they occur, as a rule, in spongy lung tissue; but in other cases they are quite **resonant,** and convey an impression to the ear of being all possessed of a definite pitch. There are only two conditions in which resonant râles occur—either consolidation exists, or there is a cavity of sufficient size to act as a resonator for râles which are produced either in itself or in a neighbouring bronchus.

The highest degrees of resonance are known as **metallic and tinkling consonances.** Here the râles have a very distinct high pitch, and give the impression of a shower of drops falling into a metallic vessel, which reverberates the sound of their fall. This is associated with amphoric breathing, and, like it, suggests either a large cavity or pneumothorax.

The position where râles are heard greatly influences the importance to be attached to their presence. If heard at the apex, they at once suggest phthisis; whilst medium and coarse crepitation at the bases may be due merely to a transient exudation which will rapidly disappear. When the patient has been breathing quietly for some hours, and especially if he has been lying in bed, a few crepitations, even if heard at the apex, may be due to temporary causes, though they should always be regarded with a degree of suspicion.

The commonest accompaniment arising in the pleural cavity is a **friction sound** characteristic of pleurisy at the stage where exudation is not abundant enough to separate the inflamed and roughened surfaces. It possesses a creaking or rubbing character, often *quite* characteristic; but sometimes, when less well marked, rather hard to distinguish from a râle. The friction sound may be fine, medium, or coarse. In some instances it is palpable, but, since coarse râles may be so too, this does not serve to distinguish them.

The chief features of difference are that friction sounds occur during that part of inspiration when the roughened surfaces are rubbing against each other, to reappear at a corresponding period of expiration. They are, moreover, unchanged after the patient has coughed, whilst râles may alter under these conditions because of changes in the disposition of the secretion which causes them. The fact that friction is sometimes more localised than crepitation may also be of service. Sometimes friction is markedly intensified by increasing the pressure with which the stethoscope is applied. This acts by causing the roughened surfaces to rub against each other more firmly. Pressure does not affect the intensity of râles. The situation of the doubtful sound, or the presence of pain, or some point in the history of the case, may assist the observer in arriving at the diagnosis.

It must never be forgotten that the presence of one form of accompaniment does not exclude the others. Any two or all three may be found coexisting in one case. When pleuritic friction is developed along the anterior edge of the left lung, and especially when that part of it which is in relation to the apical segment of the heart is affected, the friction sounds often assume the rhythm of the heart beat rather than that of the respiratory movements. Hence the sound is liable to be mistaken for pericardial friction. To distinguish between this so-called *pleuro-pericardial friction* and that of true pericarditis will rarely be very difficult if it is recollected that the former, depending as it does on the apposition of two roughened patches of pleura, is only heard during those phases of respiration when the patches are in contact. Hence a deep inspiration, by removing one of them from the other, may prevent the production of the sound, whilst in other cases holding the breath, or emptying the lungs as completely as possible, may lead to a like result. In short, pleuro-pericardial friction is much more dependent than true pericardial friction on the movements of respiration.

Hippocratic succussion is the name given to a splashing sound which can be heard when a patient who has both gas and fluid (usually pus) in the pleural cavity is shaken or moves suddenly.

SECTION VI.—PHYSICAL SIGNS OF THE PRINCIPAL PULMONARY DISEASES.

1. **Acute bronchitis.**—The patient is somewhat breathless, and coughs. The sputum is at first mucous and scanty, but subsequently becomes mucopurulent and abundant. On percussion the resonance is normal; on auscultation the breath sounds are vesicular, and accompanied by sonorous and sibilant rhonchi, the latter being especially prominent when the smaller tubes are implicated. The vocal resonance is unaltered.

2. **Chronic bronchitis.**—The signs resemble those of acute bronchitis, but pain is less, and dyspnœa is more marked. The sputum is abundant and mucopurulent. Coarse crepitations are usually abundant.

3. **Emphysema.**—The patient suffers from breathlessness, and is often somewhat cyanosed. He generally has a good deal of cough and some expectoration. The chest is barrel-shaped, and its expansion during inspiration is insufficient, whilst the expiratory phase of respiration is prolonged. On percussion there is a hyper-resonance, sometimes a trace

of tympanicity. The borders of the lungs encroach on surrounding organs, and the area of superficial cardiac dulness may be greatly lessened. Auscultation reveals weak breath-sounds and diminution of vocal resonance. This disease is often complicated by chronic bronchitis, and then the breath-sounds become harsher than normal, and there is considerable prolongation of the expiratory murmur.

4. **Phthisis.**—The earliest signs of the disease are often loss of weight and appetite, cough, and tendency to sweating during the night. At a later stage one finds severe cough, especially in the morning, sometimes hæmoptysis, increased rate of respiration, diarrhœa, hectic fever, and the other symptoms of an acute inflammatory disease. Inspection reveals in many cases a phthinoid chest with local retraction and defective movement. By palpation one detects increased vocal fremitus; by percussion, localised dulness, especially above or below the clavicle, and sometimes the physical signs of a cavity are apparent. By auscultation one finds that expiration is prolonged, or that the breathing is bronchial in character. The breath-sounds are accompanied by crepitations, most of which are of medium size. The phenomena of percussion and auscultation over the apex of the right lung are frequently equivocal: one is only justified in diagnosing phthisis there when the physical signs are very well marked. The sputum contains tubercle bacilli, and often also elastic tissue.

5. **Lobar pneumonia** is recognised by its sudden onset, with rigors, cough, and pain in the side, associated with fever, which remains continuously high. The face is flushed, the breathing rapid, and the sputum, which is not copious, is rust-coloured and excessively tenacious. The microscope reveals the presence of pneumococci. The physical signs vary with the stage of the disease. First stage: Percussion-

sound rather tympanitic but slightly dull, fine crepitations present. Second stage: Absolute dulness on percussion, high-pitched bronchial breathing, increased vocal resonance and fremitus. Third stage: Gradual diminution of dulness, disappearance of bronchial breathing, presence of medium and some fine crepitations, vocal resonance and fremitus return to normal.

6. **Chronic interstitial pneumonia.**—The patient complains of some breathlessness on exertion, and of cough with rather copious mucopurulent expectoration. The sputum may be fetid, or, when the disease is a pneumoconiosis, may contain characteristic elements. The physical signs are a gradually developed flattening over the affected region, where also expansion is absent, or defective and delayed; the shoulder of the diseased side droops. On percussion there is a dull area surrounded by one where the resonance is boxy, and the heart is drawn over by the contracted lung. Auscultation reveals feeble or bronchial breathing, and a few crepitations and rhonchi. The vocal resonance and fremitus are exaggerated.

7. **Pleurisy** is characterised by the presence of fever, pain in the side, restrained but rapid breathing, and suppressed dry cough. In the earliest stage fine friction is audible before any other abnormal signs can be detected. Subsequently, as fluid gathers, the affected area becomes dull, whilst the lung above yields a tympanitic resonance. As the dulness increases the breath sounds, vocal resonance, and fremitus diminish in intensity, and at last they wholly disappear. Above the level of the fluid, during the height of the disease, the breathing may be somewhat bronchial and accompanied by fine crepitations. As the fluid is re-absorbed in process of recovery, the dulness again decreases, whilst the breath-sounds and

vocal resonance gradually return, and when the fluid has almost disappeared friction is once more heard, but now of a much coarser character than at first. In certain cases, for some time during the advance and again during the recession of the disease, ægophony occurs. In massive effusions the neighbouring organs are displaced, and there may be bulging of the intercostal spaces.

8. **Pneumothorax.**—The patient complains of sudden pain and breathlessness. The affected side is distended and immobile, or lags behind the other. On percussion there is a loud, deep resonance more or less tympanitic, and by coin-percussion a characteristic ringing sound is elicited. The breath-sounds and vocal resonance are absent, or, if a bronchus communicates with the pneumothorax, are replaced by amphoric breathing and resonance, whilst if fluid is present one may hear metallic tinkling and elicit Hippocratic succussion. The surrounding viscera are displaced.

9. **Hæmorrhagic infarction** of the lung occurs in the course of valvular heart disease, and is characterised by the sudden onset of pain, associated with blood-stained expectoration. If the infarct is near an accessible portion of the surface of the lung, one can discover a patch of dulness, with altered breath-sounds and crepitations.

10. **Asthma** may usually be regarded as a symptom rather than a disease. Cardiac asthma has already been described, and asthmatic conditions may likewise arise from polypi or other sources of reflex nasal irritation, or from disease of the stomach or kidneys. The form known as bronchial or "spasmodic" asthma results mainly from spasm of the muscles of the smaller bronchi. In it the patient is found sitting up or leaning forward with the hands fixed on some object in order to give additional

purchase to the accessory muscles of respiration. The face is flushed and the vessels are turgid. Expiration is prolonged and laboured, and the lungs are too full of air. Percussion yields a somewhat hyper-resonant sound. Auscultation reveals at first musical râles and wheezing sounds, with marked prolongation of expiration. After secretion has been established, deeper rhonchi become audible. The sputum, which is scanty, contains small lumps, in which Curschmann's spirals and Charcot-Leyden crystals are found.

11. **Signs of pressure on a main bronchus.**—The signs in this condition are not uniform, but the following are fairly typical: There is no marked dyspnœa until an advanced stage of the disease, except after a severe paroxysm of cough, but the rate of respiration is usually somewhat increased. The patient often suffers from spasmodic cough, caused by the difficulty of forcing tough sputum through the stricture. On examining the thorax one finds that the *sound side* is expanded, and shows a large respiratory excursion and low position of the diaphragm, whilst the breath-sounds are vesicular, but harsher than usual. On the *affected side* there is usually some general retraction. Mobility is limited or wholly absent. Inspiratory retraction of the intercostal spaces often occurs. A peculiar respiratory vibration is sometimes felt near the constricted part of the bronchus, and vocal fremitus is diminished over the whole side of the chest. On percussion the resonance is normal in the earlier stages, but as the access of air becomes more obstructed the note rises in pitch. Eventually dull areas due to tumour or pleurisy may appear. On auscultation the breath-sounds are at first harsh and blowing, especially in the upper interscapular region; subsequently they generally grow weak, but a rough respiratory murmur is audible *near* the point of stenosis. Finally, the

breath-sounds are wholly extinguished, and a deep, sonorous rhonchus, best heard in the interscapular region, alone remains. Vocal resonance is diminished, and may be almost ægophonic. The changes in the lung may affect neighbouring viscera, and the heart may be drawn towards the affected side by the retraction of the airless lung, or thrust from it by the growth of an aneurysm or malignant tumour. Pain is less common than a sense of localised oppression.

SECTION VII.—THE SPUTUM.

The characters of the cough have already been treated of in a previous chapter (Chapter II.). It remains to add a few notes on the appearance and examination of the sputum in different diseases.

The following are the principal points to be observed by the naked eye :—

1. Quantity ;
2. Consistency ;
3. Whether homogeneous or in layers of different appearance ;
4. Whether frothy or airless ;
5. Colour and transparency ;
6. Odour.

The above qualities depend on the character of the material which is coughed up. The main varieties are mucous sputum, serous sputum, fibrinous sputum, purulent sputum, and blood. In many instances transition types between them are observed.

Mucous sputum is characteristically present in early bronchitis. It is clear, tough, and sticky. As a rule, the amount is not great. At a later stage of bronchitis the mucus is mixed with pus cells. The sputum is then less tough, more copious, and has a greenish yellow colour.

Muco-purulent sputum occurs in many diseases of the lung. In phthisis with cavity formation one often finds small ragged lumps of muco-pus, surrounded by mucus, which are heavier than the other constituents since they are airless. They therefore sink to the bottom and become more or less flat and buttonlike. This constitutes the "**nummular**" sputum of phthisis. If there be a fair amount of serous or watery fluid mixed with such sputum it gradually settles into three layers, the lowest being purulent, the next serous, and the uppermost composed of frothy mucus.

Sputum composed of pus alone usually proceeds from an abscess which has ruptured into the lung or air-passages.

Serous sputum occurs apart from mucous expectoration as a thin, watery fluid, generally bloodstained. It indicates œdema of the lung. Pulmonary œdema without extravasation of blood yields a white frothy sputum like soapy water.

Blood may be coughed up alone, or the sputum may be more or less bloodstained. It must be distinguished from blood brought into the mouth from epistaxis, gastric hæmorrhage, or bleeding from varicose veins in the walls of the œsophagus. Its brighter colour and its frothy appearance often make the discrimination perfectly simple. When it comes from the lungs its presence may result either from pulmonary or cardiac disease, or from aneurysm.

Several diseases cause a **characteristic coloration of the sputum.** Thus, in pneumonia it is **rusty** and so viscid that it often will not fall out of an inverted spittoon; it is **bright yellow or green** when a liver abscess has ruptured into the lung, and the latter colour also appears in some cases of pneumonia. Sometimes when an amœbic hepatic abscess has discharged by the lung, the sputum has the appearance of **anchovy sauce.** **Black spu-**

tum is common with coal miners, whilst red-streaked sputum is suggestive of phthisis. **Prune-juice sputum** occurs when blood lingers in a lung which has become œdematous. Thus it is found in cases of chronic pneumonia that are going on to disintegration of the lung tissue. **Red-currant-jelly sputum** is said to be characteristic of malignant disease in the lung. It has also been found in hysteria.

The **quantity** of sputum coughed up in twenty-four hours is important; and still more so whether large quantities are rapidly got rid of at considerable intervals or whether it comes away in small amounts and frequently.

Occasionally small **casts of bronchi** are to be found in the sputum, but the examination for formed elements is best conducted with the aid of a microscope.

The **odour of the sputum** is seldom very characteristic. Ordinarily it has a "stale" smell, but in cases of gangrene of the lungs, of fœtid bronchitis, and of bronchiectasis it may develop an exceedingly penetrating putrid odour. An unpleasant odour may also be acquired during its transit through the mouth.

Microscopic examination of sputum.—Generally it is well first to examine an unstained and fresh specimen, and thereafter to use special methods for the recognition of bacteria. To select a suitable piece, place the sputum in a flat glass vessel, which can be laid on either a white or a black background as is found convenient. Mixed with the amorphous mucous exudation which forms the basis of the sputum may be seen various organised and crystalline substances, of which the following are the principal groups:—

I. **Cellular structures.**—(1) **Pus corpuscles** in various stages of granular degeneration and with several nuclei.

(2) **Epithelium** from mouth, air-passages, and alveoli. The latter may contain pigment which has reached them from the air, or they may exhibit a very characteristic iron-containing pigment, which is unusually abundant in cases of heart disease with pulmonary congestion, and indicates brown induration of the lung. This pigment yields the hæmosiderin reaction on the addition of hydrochloric acid and potassium ferro-cyanide.

(3) **Salivary corpuscles** are picked up by the sputum in its passage through the mouth.

(4) **Red blood corpuscles.**—A few are of no importance. Large numbers occur in hæmoptysis.

(5) **Eosinophil cells** occur in asthma, and are often associated with Charcot-Leyden crystals. They are large, and contain numerous fine granules which stain with eosin.

II. **Elastic fibres** indicate destruction of lung tissue, whether from phthisis, gangrene, or abscess. In gangrene only a few fibres escape the destructive process. They are found in the small tough lumps of the sputum, and are best demonstrated by a rapid heating with an equal quantity of 10 per cent. solution of caustic soda, which liquefies the other elements more quickly than these fibres. After boiling, a gelatinous mass is left, to which a considerable quantity of water should be added and the mixture left in a conical glass till the elastic fibres settle to the bottom. Thus they may be isolated, and in well-marked cases exhibit the alveolar arrangement of the lung tissue. Too prolonged an exposure to the caustic will lead to the solution of the elastic fibres as well as of the other constituents (Fig. 80).

III. **Fibrin casts,** often large enough to attract the unaided eye, are still more frequently visible under a low power of the microscope (Fig. 81).

IV. **Curschmann's spirals** are found in the sputum of asthmatic patients. Some of the sputum should be spread out on a piece of glass on a black surface. The spirals look like little sago grains. When unrolled they appear as convoluted threads which may be quite an inch in length. Under the microscope they show a central core, round which a sheath of tough mucus, with a large number of small round cellular elements in it, is coiled (Fig. 82).

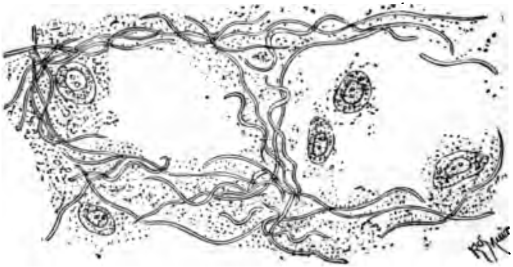


Fig. 80.—Elastic tissue from lung in sputum of a case of phthisis. × 300.

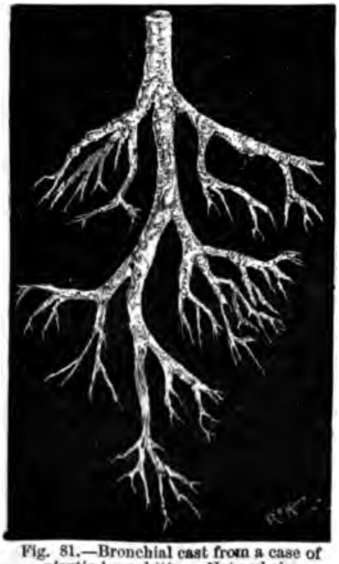


Fig. 81.—Bronchial cast from a case of *plastic bronchitis*. Natural size.

V. **Crystals.**—(1) In asthma, fine colourless crystals with sharp extremities are often found. They are frequently associated with the spirals already described, and are known as **Charcot-Leyden crystals.** They are probably phosphates of an organic base (Fig. 83).

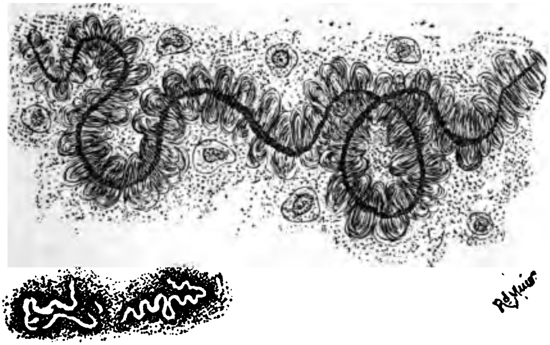


Fig. 82.—Curschmann's spirals in sputum. × 200 and natural size.

(2) **Fatty acid crystals** are needle-shaped, and generally occur in clusters.

(3) **Cholesterin** occurs in rhomboidal plates, which generally have a small notch in one corner. They occur in old purulent sputum from pulmonary cavities, but their presence is uncommon.

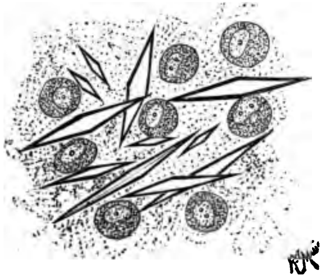


Fig. 83.—Charcot-Leyden crystals. × 350.

(4) **Hæmatoidin crystals** occur where there has been an old hæmorrhage in cases of abscess and empyema. They have a characteristic

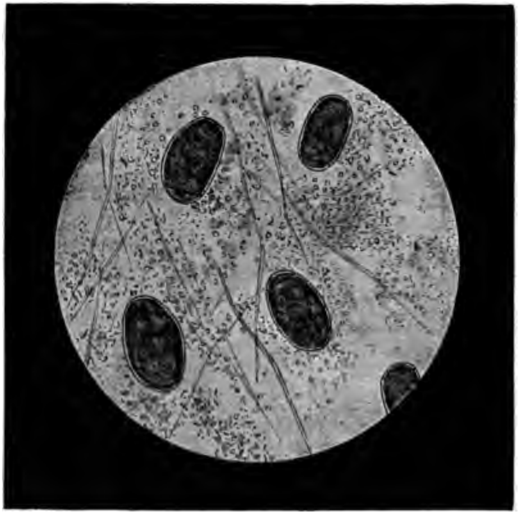


Fig. 84.—Ova of Distoma pulmonale in sputum. High power.

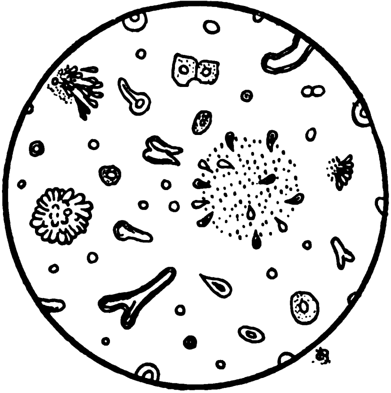


Fig. 85.—Actinomyces in sputum.

brown-yellow colour, and appear as needles, rhombi and plates.

(5) **Leucin and tyrosin** may be found on rare occasions in pus from old perforated empyemata.

VI. **Parasites.**—These belong both to the animal and vegetable kingdoms. Of animal parasites **echinococci** are the most important. The presence of hooklets, and still oftener of fragments of the laminated ectocyst, are the usual indications of their existence (*see* p. 103). In China and other parts of Eastern Asia **Distoma pulmonale** is frequently found. Its presence causes sharp attacks of hæmoptysis, which may be mistaken for the hæmoptysis of phthisis. Microscopic scrutiny of the sputum, however, *generally* reveals characteristic ova (Fig. 84).

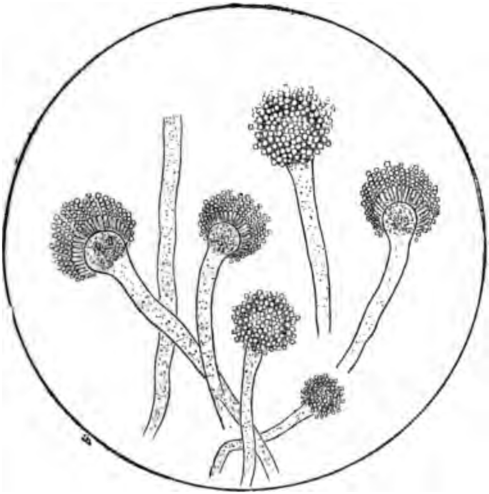


Fig. 86.—Aspergillus fumigatus. × 300.

The vegetable parasites are fairly numerous. Besides bacteria, which are considered in a separate chapter, and amongst which the most important are tubercle bacilli, pneumococci, and Pfeiffer's bacillus, some of the higher fungi are also found, the most important being **actinomyces** and **aspergillus fumigatus** (Figs. 85, 86).

APPENDIX TO CHAPTER VI.

ON GRAPHIC METHODS OF RECORDING THE CONDITIONS OBSERVED IN THE HEART AND LUNGS.

1. **Full-size outlines of the heart.**

The position of the nipples, and the outlines of the clavicles, ribs, and sternum, should be carefully traced on the chest with a dermatograph pencil, and the same should be done for the outlines of relative and absolute dulness of the heart and liver. These tracings should then be gone over rapidly with a small paint brush dipped in sweet almond oil, and a sheet of tissue paper pressed down upon the patient's chest. The oil will leave a mark on the paper, which can be more strongly traced by a pencil after the paper has been removed.

An alternative method is to **photograph the chest** with the lines drawn in. This has the convenience of preserving the record in a less bulky form. A scale of inches should in this case be laid across the patient's epigastrium to permit of absolute measurements being taken from the photograph.

2. **The use of symbols on outline charts.**

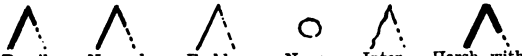
(*a*) **The heart.**—The presence and position of murmurs are easily indicated by shading. Their intensity is roughly represented by the heaviness of the shading. Examples of this method are seen in Figs. 37, 38, and 43. When two synchronous murmurs are present, and one wishes to show where the first, after becoming fainter, gives place to the second, which grows increasingly loud as the stethoscope is carried along the line joining their areas of maximum intensity, one makes use of the musical signs of diminuendo and crescendo, **>** and **<**. The situation of pericardial friction is indicated by a zig-zag line, ∧∧∧∧. When the apex beat does not reach the edge of the deep cardiac dulness, its situation is shown by a small cross, ×.

(*b* **The lungs.**—The position of any dull area is indicated by shading. If, following the suggestion of Prof.

Sahli, one represents superficial dulness in blue chalk and deep in red, one can not only make the record clearer, but can also superpose on the same chart auscultatory phenomena in black symbols without clogging it. The auscultatory phenomena are well represented by the following symbols* :—

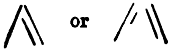
1. Types of breathing :—

(*a*) Vesicular.

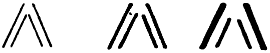


Puerile. Normal Adult. Feeble. None. Interrupted. Harsh, with expiration prolonged.

(*b*) Transition Type. } "Bronchovesicular" or indeterminate

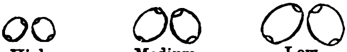


(*c*) Bronchial.



Tubular or High-pitched. Medium-pitched. Low-pitched.

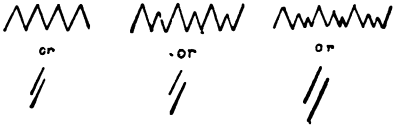
(*d*) Amphoric.



High-pitched. Medium-pitched. Low-pitched.

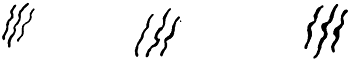
2. Accompaniments :—

(*a*) Friction.



f Fine. *m* Medium. c Coarse.

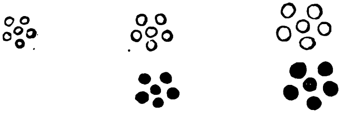
(*b*) Dry râles.



Sibilant rhonchi. Medium-pitched rhonchi. Sonorous rhonchi.

(*c*) Moist râles :—

{ Non-consonating.
{ Consonating.



Fine. Medium. Coarse.

* Mostly after Professor Wyllie.

If the accompaniments occur during inspiration, the letter *i* is prefixed to the symbol; if during expiration, *e*.

Vocal resonance is indicated by the letters V.R., followed by + 1, + 2, + 3, if it is slightly, moderately, or greatly increased; or − 1, − 2, − 3, if it is proportionately diminished.

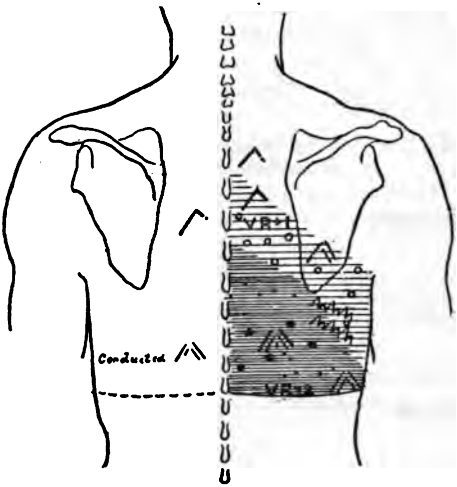


Fig. 87.—Record of pneumonia.

Other physical signs are best indicated by various letters of the alphabet. The accompanying figure (Fig. 87) illustrates the application of these symbols to a case of pneumonia.

CHAPTER VII.

THE URINE.

THE method of interrogating a patient whose symptoms point to an affection of the urinary system has already been described (p. 9), and the physical examination of the kidneys has been considered along with that of the other abdominal organs (p. 78).

In this chapter we propose to take up the examination of the renal secretion.

Collection of Samples.

Owing to the variations in the composition of the urine at different times of the day, the sample examined should, if possible, be taken from the total urine of the twenty-four hours. If only one sample can be obtained it should be that which is passed about three hours after taking a meal, as abnormal ingredients are then more likely to be present. The sample should be poured into a tall conical glass, covered, and allowed to stand for some hours in a cool place. If it be desired to preserve the urine for some time, two or three drops of formol should be added.

Any suspended matters soon settle to the bottom of the glass, and the examination of the sample may then be proceeded with. This should be conducted (1) physically, (2) chemically, (3) microscopically.

SECTION I.—PHYSICAL EXAMINATION OF THE URINE.

Attention should be paid to the following points: (*a*) Quantity, (*b*) colour, (*c*) consistence, (*d*) odour, (*e*) density, (*f*) naked-eye characters of the deposit.

(*a*) **Quantity.**—The amount of urine passed during the day should be measured separately from that passed during the night. The sum of the two gives the total for twenty-four hours. The bladder should be emptied at a fixed hour—say 8.30 a m., and the product discarded. All the urine passed during the day is carefully collected, and the bladder emptied again at 8.30 p.m., the product being added to the day's secretion. This is the amount of the "*day urine.*"

The bladder is again emptied at 8.30 next morning, and the product added to that which has been passed during the night. The total quantity is the "*night urine.*" This added to the day urine gives the total for twenty-four hours.

It is often difficult to collect all the urine that is passed, some being lost with the motions. This is especially the case with children, female patients, and those who pass their evacuations involuntarily. Where great accuracy is required, recourse must be had to the catheter.

A healthy adult male passes on an average 50 ozs. (1450 cc.) of urine in twenty-four hours; women a few ounces less.

The following table represents the amount of urine passed daily by children of different ages (Holt)* :—

---

* Churchill, in a paper read before the American Pediatric Society in 1898, asserts, as the result of his own observations, that the amount of urine passed by children is less than is usually supposed. He gives the following averages of quantity and specific gravity at different ages :—

Age.	*Quantity.*	*Specific Gravity.*
3 years.	358 cc.	1024
4 ,,	299 ,,	1027
5 ,,	392 ,,	1024
6 ,,	405 ,,	1023
7 ,,	564 ,,	1018
8 ,,	628 ,,	1021
9 ,,	731 ,,	1020
10 ,,	768 ,,	1023
11 ,,	716 ,,	1018
12 ,,	829 ,,	1021

See also "An Essay on Clinical Urology in Infancy and Childhood," by Fernandes Figueira, *Lancet*, Sept. 12th, 1896.

First 24 hours	0 – 2	ozs.
Second 24 hours	$\frac{1}{3}$ – 3	„
Three to six days	3 – 8	„
One week to two months	5–13	„
Two to six months	7–16	„
Six months to two years	8–20	„
Two to five years	16–26	„
Five to eight years	20–40	„
Eight to fourteen years	32–48	„

Above the age of 15 the quantity passed is about up to the adult standard.

It will be observed that, relatively to their weight, children pass more urine than adults. This is to be attributed to the relatively greater activity of the metabolic processes in children and to the more fluid nature of their diet. The above quantities, however, are only roughly approximate, and in many cases one will find that the amount of urine excreted by a child of given age is smaller than the quantity tabulated.

Normally, very much more urine is secreted during the day than during the night. The normal proportion of day urine to night urine is : : 100 : 25–60. Approximation of the night quantity to that of the day is always abnormal, and is especially apt to occur in chronic renal disease, of which, indeed, it may constitute one of the earliest signs. Thus the proportion of day to night urine may become : : 100 : 100 or even 200. The solids are increased in proportion to the water.

An *increased secretion* of urine occurs physiologically after increased consumption of food or drink, and after exposure to cold. Conversely, one finds the *secretion diminished* when little food or drink has been taken, and after exposure to heat—especially if followed by sweating.

A *pathological increase* in the urine occurs in diseases associated with an increased arterial pressure —*e.g.* granular kidneys—also in both forms of diabetes, during the absorption of exudates and in some neurotic conditions—*e.g.* hysteria. *Abnormal diminution* of urine is found where the arterial pressure is lowered or the intravenous pressure in the kidney increased—*e.g.* in advanced mitral disease; also in all fevers and in cerebral irritation—*e.g.* concussion.

(*b*) **Colour of the urine.**

Normal urine is said to have the colour of amber or pale sherry. The exact tint fluctuates widely even in health, depending upon the degree of dilution and upon the reaction. An acid urine is always darker than one which is alkaline, even when they are equally concentrated. The colour of normal urine is due to a yellow pigment, to which the name of urochrome has been given. Urobilin only occurs in very small quantity in the urine under normal conditions. In febrile and some other diseases, however, a large quantity of urobilin may appear in the urine. The latter has then a warm orange colour, and usually shows a dull pink tint at the apex of a conical glass.

If examined spectroscopically* in a thin layer the urobilin band will be seen in the green between

---

* The following directions for the spectroscopic examination of urine are given by A. E. Garrod:—

1. Use a small direct vision spectroscope.
2. Examine the urine in a 6 oz. conical glass. This permits of the inspection of layers of different thickness.
3. Hold the slit one inch from the glass, and move it up and down the entire length of the cone.
4. Either daylight or artificial light may be employed.
5. If in doubt as to the bands, shake up 400 cc. of the urine with 50 or 60 cc. pure amylic alcohol and a few drops of acetic acid. Collect the layer which floats. Clear it if need be by the addition of a little ethyl alcohol, filter, and examine.

b and F (Fig. 88). Such a urine is often dichroic—looking red by transmitted and green by reflected light. The presence of excess of urobilin may be confirmed by rendering the urine strongly alkaline with ammonia, filtering, and adding to the filtrate a few drops of a 10 per cent. solution of chloride of zinc. If excess of urobilin is present, the solution becomes fluorescent.

The following are the chief varieties of alteration in colour of the urine, with their causes :—

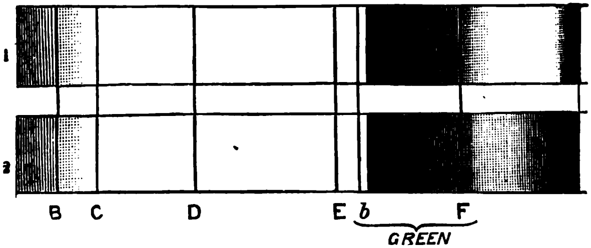


Fig. 88.
1, Spectrum of Urobilin.
2, Spectrum of Urobilin masked by other pigments.

Pallor.—This is present whenever a large excess of urine is being secreted. It may also occur from an absolute diminution in the amount of urinary pigment. This is the case in diabetes; a diabetic urine is pale even when concentrated.

Orange-coloured or reddish brown.—After the administration of rhubarb, senna, and chrysophanic acid. Distinguished from blood by the fact that addition of a mineral acid causes the urine to become yellow, while an alkali turns it dark red. Some bilious urines are also of an orange tint.

Dark brown.—This may be due to methæmoglobin, which is found in hæmorrhage into the kidneys, and in paroxysmal hæmoglobinuria. The spectroscope shows a band in the red in addition to two bands very near those of oxyhæmoglobin (p. 232).

Red.—From blood (*see* p. 336), or from aniline dyes contained in sweets.

Port-wine coloured.—Due to the presence of hæmatoporphyrin (*see* p. 338).

Brownish black.—This may be due to the presence of melanin, which is sometimes excreted in the urine in cases of extensive melanotic sarcoma wherever situated. Such a urine usually darkens on standing, and may even become quite black. It does not reduce cupric oxide in alkaline solution. On addition of ferric chloride the urine yields a brownish turbidity, or even a black precipitate which is soluble in excess.

Greenish black.—From the presence of aromatic compounds (hydroquinone, etc.) after the administration of carbolic acid, guaiacol, salol, resorcin, naphthaline, etc.

Greenish or yellowish green.—From the presence of bile (*see* p. 353). The administration of santonin is also followed by a yellowish green colour, but on adding an alkali the urine becomes dark red.

Yellowish and milky.—From the presence of pus (*see* p. 356) or of fat. The fat may form an emulsion as in chyluria, or it may be present in the form of droplets which float on the surface of the urine (lipuria); the latter condition sometimes occurs in advanced fatty disease of the kidney. The presence of fat in the urine can always be shown by the addition of a little caustic soda and ether. On shaking up the mixture the fat is dissolved out, and is left behind on evaporation of the ether.

Bluish or greenish blue.—This occurs when the urine contains a large excess of indigo-forming substances—*e.g.* in some cases of typhus. It is especially seen after putrefaction has occurred.

The urine may be **opalescent** from the presence of various substances in suspension. If the opalescence persists after filtration, it is due to the presence of bacteria.

A slight opalescence which causes the urine to look smoky is produced by the presence of small quantities of blood (*see* p. 336). •

Alkaptonuria.*—This is a condition in which X the urine is natural-looking when passed, but when exposed to the air it becomes gradually darker from the surface downwards; ultimately it may be dark brown or black. It is due to the presence in the urine of di-hydroxyphenyl acetic (homogentisinic) acid.

2. Does not polarize –

It may occur spontaneously in quite healthy persons. The addition of an alkali causes the urine to become dark at once. Such urines reduce alkaline solution of cupric oxide, but the bismuth test for sugar is negative. Urines containing melanin also become darker on exposure to the air owing to oxidation of the pigment, but they do not reduce cupric oxide. Vide Baumann Hoppe Seyler - p. 460 for [illegible]

Carbolic acid urine also becomes darker on exposure to air owing to the oxidation of the hydroquinone which it contains into pigments not yet fully investigated.

In alkaline urines an **iridescent pellicle** frequently appears on the surface. When the urine has cooled this can be skimmed off like a thin brittle film. It is composed of phosphate of lime. The idea formerly entertained that such a pellicle occurs especially in the urine of pregnancy is groundless.

(*c*) **Consistence of urine.**—In health the urine is quite watery in consistence. If much sugar or bile is present it is less mobile, and in the presence of bile or of much albumin the froth which forms on shaking is more persistent than is usual. Alkaline urine containing pus may be quite ropy. A special alteration in consistence occurs in the condition known as **Fibrinuria.** When this is present the urine is reddish yellow when passed, but soon sets into a jelly, which contracts somewhat on standing. If only little fibrin is present the whole urine may not coagulate, but a sticky sediment forms at the bottom of the vessel. Fibrinuria is due to the entrance of blood plasma into the urinary tract. It occurs as a very rare symptom of villous growth in the bladder, and sometimes also after the administration of cantharides. The fibrin may be recognised as such by washing it in 5 or 10 per cent. salt solution, with the addition of a little thymol, and then placing in 1 per cent. HCl. It swells up, but is not dissolved unless pepsin be also added.

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* Alkapton = Alkali and κάπτειν, from the affinity of the compound *for alkali*.

(*d*) **Odour.**—Normal urine has a characteristic "aromatic" odour. When the urine has stood for some time the odour becomes ammoniacal. In cases where there is an abnormal communication between some part of the urinary tract and the intestine the odour may become fæcal. In acetonuria the odour is fruity. After the administration of turpentine the urine has an odour like violets. Cubebs, santonin, and some other drugs also impart to it their peculiar smells. In diabetes the odour has been compared to that of new-mown hay.

(*e*) **Density.**—Clinically the specific gravity of urine is always taken with the instrument known as a **urinometer.** An ordinary urinometer is graduated for a temperature of 15° C., and will record variations in specific gravity from 1000 up to 1060.

How to use the urinometer.—The urine should be allowed to cool, and should be placed in a tall jar, wide enough to allow the urinometer to float freely without touching the sides. All bubbles must be removed from the surface by means of bibulous paper. The urinometer should be wiped clean and placed floating in the centre of the jar. The eye is then placed level with the surface of the urine and the division of the scale to which the latter reaches read off. Care must be taken to read the level of the true surface of the urine, not the edge of the rim which heaps itself up around the shaft of the urinometer.

If only a small specimen of the urine is obtainable it may be necessary either to use "specific gravity beads," or else to add water to it in order to get enough fluid to float the urinometer. The specific gravity found is then multiplied by the necessary figure according to the degree of dilution.

Normal urine has a specific gravity varying from 1015 to 1025. If very concentrated the specific gravity may rise to 1035 even in health. During the first month of life the specific gravity varies between 1001 and 1005, but by the second year it has reached 1026 or 1030, and in older children the urine tends to be rather more concentrated than that of adults.

In normal urine the specific gravity is in direct proportion to the amount of urea present. An abundant urine of *low* specific gravity is suggestive either of diabetes insipidus, or of chronic renal disease. An abundant urine of *high* specific gravity is characteristic of diabetes mellitus. In the latter condition the specific gravity may reach 1075; in most cases, however, it is between 1040 and 1045. In diabetes insipidus, on the other hand, the specific gravity may fall to nearly that of distilled water.

The presence of albumin in the urine does not materially affect its specific gravity. It should also be borne in mind that the urinometer is entirely unaffected by the presence of merely suspended substances.

Estimation of the amount of solids.—This may be roughly done by multiplying the last two figures of the specific gravity by 2·33. The result is the number of grammes of solids in 1 litre of the urine, or the number of grains of solids in 1,000 fluid grains of urine; *e.g.* if the specific gravity of a urine is 1020, it contains 20 × 2·33 = 46·6 grammes of solids in every litre, or 4·6 per cent. This multiplied by 4·375 gives grains per ounce—in this case 20·1. The average daily output of solids in the urine is about 60 to 70 grammes (2–2½ oz.). The above mode of calculation is not applicable to urine containing abnormal ingredients, *e.g.* sugar or albumin.

(*f*) **Naked-eye characters of the deposit.**—When normal urine has stood for some time there appears in it a deposit of "**mucus.**" This forms a woolly-looking cloud which usually settles to the bottom of the glass, but, if the urine be of high specific gravity, may be in the middle of the glass or even at the top. It should be mentioned that this substance probably does not consist of true mucin. It has been described as a nucleo-albumin. This, however, is by no means certain, as the body has not been investigated with regard to the presence or absence of the different essential characters of the nucleo-albumin group.

If traces of blood are present in the urine the cloud of "mucus" has often a brownish tint.

The normal urinary ingredients which may separate out in the form of a deposit, visible to the naked eye, are—earthy phosphates, urates, and free *uric acid.*

Phosphates.—The phosphates of calcium and magnesium separate out if the urine is neutral or alkaline. They form a colourless deposit. It can be recognised by the fact that if a little of it be transferred by a pipette to a test-tube, and some dilute acetic acid added, the deposit dissolves. A deposit of **pus** is apt to be mistaken for one of phosphates. They may be distinguished by moving the glass gently from side to side. It will then be found that a deposit of phosphates is more flocculent and less compact than one of pus, and the surface layers of it are easily detached on shaking, and float up; this does not happen in the case of pus. Acetic acid also does not dissolve pus, while the addition of caustic alkali renders it ropy. If the urine be acid, however, and its reaction has not been tested, a deposit of pus cannot be distinguished with the naked eye from a deposit of phosphates. It should also be borne in mind that deposits of phosphates and pus often occur together.

Urates.—The *urates* of sodium, potassium, and ammonium may form a deposit if the urine be concentrated or highly acid. They may appear, even in health, when the urine cools. Owing to their affinity for the urinary pigments the deposit is usually coloured, being commonly red, or like terra-cotta, form ing what is known as the **"brick-dust" deposit.** If the urinary pigment be scanty, however, the deposit may be merely yellowish, or even colourless. Deposits of urates can always be recognised by the fact that they disappear rapidly on heating the urine. The heating ought to be accomplished gradually, because the urine might also contain albumin, which, if the urine were rapidly heated, might be coagulated before the deposit of urates had all had time to clear up, and thus confusion might arise. Acetic acid does not dissolve a deposit of urates. On the other hand, strong mineral acids, such as nitric acid, dissolve the deposit at once with the production of effervescence.

Acid urate of soda is a rare deposit. It occurs in acid urines. It forms a yellowish, granular, sandy-looking sediment. It does not dissolve readily on heating.

Acid urate of ammonia forms a very similar deposit, but it occurs in ammoniacal urines, and is therefore mixed up with a deposit of phosphates.

Uric acid.—This may form a scanty deposit visible to the naked eye. The deposit occurs in the form of crystalline grains of a darkish brown colour, and is therefore known as the **"cayenne-pepper deposit."** When in doubt use the microscope.

The sulphates practically never form urinary deposits. **Oxalates** do, but the deposit is always scanty, mixed up with the cloud of mucus, and not easy to recognise with the naked eye. We have already spoken of the occurrence of fibrin, and the other abnormal ingredients which may be deposited will be described in the section on the microscopical examination of the urine.

We would warn the reader against the common mistake of supposing that a substance is necessarily being excreted in excess when it appears in the urine in the form of a deposit. This, of course, is not necessarily the case at all. Thus the occurrence of a "cayenne-pepper" deposit does not necessarily mean that the patient is excreting an excess of uric acid. It may merely be due to the fact that the conditions which normally cause the uric acid to be in solution have become modified. The urine may be abnormally acid, for example, or it may be deficient in colouring matter or in salts, all of which conditions tend to lessen the solubility of uric acid, and to favour its deposition in the form of crystals. Similarly in the case of a deposit of phosphates. That does not mean that more phosphoric acid is being eliminated; it merely indicates that the urine has become alkaline.

SECTION II.—CHEMICAL EXAMINATION OF THE URINE.

1. **Reaction.**—This is taken with litmus paper. The urine is usually acid in reaction. This is not due to the presence of free acid, but to acid salts—chiefly the acid phosphate of sodium (NaH_2PO_4). Sometimes the reaction is blue to red litmus paper, and red to blue litmus paper. This amphoteric reaction is due to the presence of large quantities of the disodic phosphate (Na_2HPO_4) in addition to the acid salt. It has no clinical significance. The urine may be normally alkaline after meals. This is sometimes known as the "*alkaline tide.*" It reaches its acme three hours after the taking of a meal. It is chiefly due to the disodic phosphate replacing the acid salt. Alkalinity of the urine may be due to ammonia. This can be detected by its smell, also by the fact that if the red litmus paper which has been turned blue be heated, the red colour is restored, owing to the ammonia being driven off.

The degree of acidity of the urine cannot be measured by direct titration with a standard alkaline solution in the usual way, for the reason that none of the phosphates of sodium react neutral to the usual indicators. To those who might wish to make the estimation the method of Lieblein* may be recommended, but it is too complicated for general use. In any case the exact quantitative estimation of the acidity of the urine is of no clinical service. For ordinary purposes, the intensity of the red colour produced in litmus paper is a sufficient indication of the degree of acidity.

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* *Neubauer & Vogel,* "Analyse des Harns," 10th Ed., p. 734.

2. **Examination of the urine for its normal non-nitrogenous constituents.**

(1) **Chlorides.**—Chloride of sodium is the chief inorganic constituent of normal urine. Small quantities of the potassium salt also occur.

Qualitative test for their presence.—Filter the urine if not already clear. If albumin is present, remove it by boiling. Add to ½ an inch of the urine in a test tube a few drops of nitric acid (be sure that the acid used is quite pure and free from HCl), and then as much of a 3 per cent. solution of nitrate of silver as there is of urine. If the normal amount of chlorides is present, an abundant curdy precipitate appears at once. If the chlorides are diminished, the solution merely becomes milky. If a mere trace of them is present, the solution is opalescent, and if they are altogether absent it remains quite clear.

The use of the nitric acid is to prevent the precipitation of phosphate of silver.

Quantitative estimation.—For ordinary clinical use **Mohr's method** is to be recommended. One proceeds as follows:—

Place 10 cc. of the urine, freed if necessary from albumin, in a beaker, and mixed with 50 cc. of distilled water. Add three drops of a solution of neutral chromate of potassium (1 in 20) and a pinch of calcium carbonate. The use of the latter is to neutralise any free acid that may be present. Fill a burette with standard solution of nitrate of silver (Appendix, 9). Run the silver solution into the beaker, stirring all the time. A precipitate of chloride of silver falls out. Whenever the least trace of a pink colour appears, stop. This can be best appreciated by allowing the precipitate to settle, which it very quickly does. If the sediment is in the least flesh-coloured, enough silver solution has been added. *The appearance* of the pink colour indicates that the silver has united with all the chlorides present and has begun to form chromate of silver with the potassium chromate.

Calculation.—One cc. should be deducted from the total number of cc. of silver nitrate used. The reason for this is that there exists in urine, besides chlorides, other substances with which the silver unites more readily than it does with the chromate. Roughly, the deduction of 1 cc. is sufficient to allow for these. Every remaining cc. of the solution used is equivalent to 10 milligrammes of sodium chloride. Suppose 11 cc. to have been used in all, deducting 1 cc. there is left 10 cc. This is equivalent to 100 mg. sodium chloride, which will be the quantity of chlorides in the amount (10 cc.) of urine used. If 1,500 cc. was the amount of urine, in twenty-four hours it will contain 15 grammes of sodium chloride.

For the accurate estimation of *small quantities* of chlorides in the urine the method of *Volhard* should be employed, but for a description of it special works must be consulted.

About 12 grammes represents the average daily excretion of chlorides in health. The chief cause of physioiogical variation is the nature of the diet. Pathologically, chlorides are found to be diminished in all febrile affections with the exception of malaria. In the latter disease the chlorides are increased during the febrile period, diminished in the apyrexial intervals. In acute croupous pneumonia, the chlorides are more markedly diminished than in any other fever. They may indeed disappear entirely. We regard their behaviour as of great diagnostic value. In no other disease, except, perhaps, typhus and rheumatic fevers, does such a notable diminution occur. In the diagnosis of pneumonia from empyema and pleurisy the test is of special help. The chlorides are increased after *the crisis* in pneumonia, the increase, however, not usually manifesting itself till the third day after fever had ceased, and also in cases where the rapid absorption of a large exudation is taking place.

(2) **Phosphates.**—Phosphoric acid occurs in the urine in two chief forms of combination. Combined with potassium, sodium, and ammonium, it forms the alkaline phosphates; with calcium and magnesium, the earthy phosphates. Three-fourths of the total phosphoric acid is combined with the alkalies, and only one-fourth with the earths. The alkaline phosphates, being readily soluble, never form a deposit. The earthy phosphates are insoluble in an alkaline medium, hence they are precipitated when the urine loses its acid reaction. This precipitation is aided by the action of heat. The heat probably acts by driving off carbonic acid. Hence if a urine, the reaction of which is not acid, be heated, a cloud of earthy phosphates may appear. This is distinguished from albumin by its ready disappearance on adding a few drops of acetic acid.

Qualitative tests for phosphoric acid in urine.—Place half an inch of clear urine in a test tube. Add a few drops of a solution of uranium acetate or nitrate and a little sodium acetate solution (Appendix, 11). A somewhat greenish precipitate which does not disappear on adding acetic acid indicates the presence of phosphates.

Quantitative estimation.—Fill a burette with standard solution of uranium nitrate (Appendix, 10). Measure 50 cc. of the urine to be examined into a porcelain dish or a medium-sized beaker. (If the urine is very concentrated, 20 cc. of it will be sufficient.) Add to the urine 5 cc. of an acetic acid solution of acetate of soda (Appendix, 11). If only 20 cc. urine were taken, add 2 cc. of the solution. Add also a few drops of tincture of cochineal to serve as *an indicator.* Heat the urine on a water bath. If the latter is not obtainable, use a tripod covered with wire gauze. The urine should be heated to a temperature *just short* of boiling. When heated, run in the uranium solution, stirring all the while. As a rule, 16 cc. may be run in right away. A precipitate of uranium phosphate falls down. Continue cautiously to add the uranium solution until the precipitate has a *slight but persistent greenish tint.* This is a sign that the uranium has united with all the phosphoric acid present and is beginning to re-act with the cochineal. The presence of albumin or sugar does not affect the reaction. The use of the acetate of soda in the above process is to unite with the nitric acid liberated by the union of the uranium with the phosphoric acid.

Calculation.—Suppose 20 cc. uranium solution have been required. The solution was made of such a strength that each cc. = 5 milligrammes phosphoric anhydride; 20 cc. are therefore equivalent to 0·1 gramme P_2O_5, and that is the amount in 50 cc. urine. If the patient is passing 1,500 cc. urine in twenty-four hours, his daily excretion of P_2O_5 will be 3 grms.

Normally, 2–3 grms. of phosphoric anhydride are excreted daily. Physiological variations depend chiefly upon the food. The phosphates are often considerably diminished in renal disease, but not, apparently, out of proportion to the other solids of the urine. They are said to be increased in wasting diseases of the nervous system. Their behaviour in fever is inconstant.

(3) **Sulphates.**—Sulphuric acid occurs in the urine in combination with sodium and potassium (inorganic sulphates), and with cresol, phenol, indol, skatol, pyrocatechin, etc. (organic sulphates). The former are ten times more abundant than the latter.

Test for inorganic sulphates.—Add to 10 cc. urine a few drops of hydrochloric acid and one-third of its volume of 10 per cent. barium chloride solution. If the normal amount of sulphates is present, an opaque milkiness develops. If the precipitate is thick and creamy, the sulphates are in excess; if a mere opalescence appears, they are diminished.

About 2½ grms. of sulphuric acid (SO_3) are excreted daily. The exact determination of the total sulphates, and of the proportion of inorganic to organic, is a gravimetric process unsuited for ordinary clinical work.

An approximate notion, however, of the proportion of **organic sulphates** present may be obtained by the following procedure: Add to the urine an equal volume of alkaline barium chloride solution (two parts of baryta water to one part of barium chloride solution). This precipitates the *inorganic* sulphates along with phosphates. Filter. Render the filtrate pretty strongly acid with hydrochloric acid, and heat almost to boiling. The *organic* sulphates are thus decomposed and thrown down in the inorganic form. Normally they should form merely a white cloud. If the precipitate is at all dense, the proportion of organic sulphate is in excess.

The total sulphates are increased by an increase in the diet, and in fever. The amount of sulphuric acid excreted in organic combination is increased when a larger quantity than usual of the aromatic substances with which it is combined enters the circulation. This occurs when phenol and allied substances are given as drugs, or when the production of such substances in the body is increased, as it is whenever putrefactive processes are going on. Thus the amount of organic sulphates is increased in cases where putrid abscesses have formed, or where there is retention of the intestinal contents.

(4) **Oxalates.**—Oxalic acid occurs in the urine, combined with calcium. The salt is usually kept in solution by the acid phosphate of sodium present in the urine. It is found as a precipitate, however, in about one urine out of every three. This is due to the absence of a sufficient amount of the phosphate of soda to keep it in solution. It does not necessarily mean that the excretion of oxalic acid is increased, although it is true that the more oxalic acid there is present, the greater is the tendency for it to be precipitated. About 0·017 grm. is the average amount of oxalic acid excreted daily. It is probably all derived from the food. It is increased after the taking of certain vegetables, especially cabbage, tomatoes, and rhubarb. The so-called "oxaluria" seems to be merely a variety of acid dyspepsia.

3. Examination of the urine for its normal nitrogenous constituents.

Of the total amount of nitrogen in the urine—

84–87% is in the form of urea;
2– 5% " " ammonia compounds;
1– 3% " " uric acid;
7–10% " " "extractives" (including xanthin bases).

Estimation of total nitrogen by Kjeldahl's method (modified).—Measure out 5 cc. of urine with a pipette and place it in a Kjeldahl's flask of about 150 cc. capacity, add to it 15 cc. of pure sulphuric acid and a crystal of pure sulphate of copper about the size of a split pea; heat on net till the mixture is colourless or pale green; this takes about half an hour or less; violent boiling should be avoided. Allow to cool, then dilute with 50 cc. of distilled water, again allow to cool, transfer to a distillation flask of about

700 cc. capacity, and add enough 23% solution of caustic soda to render the mixture almost neutral; add also a pinch of talc to prevent bumping. When cool add more soda (till the fluid is deep blue in colour), and close at once with the stopper connected to the condensing tube of the distillation apparatus; measure into a flask 100 cc. of a decinormal solution of oxalic acid, and let the lower end of the condensing tube just dip below the surface of this solution; then boil the blue fluid. The nitrogen passes off in the form of ammonia and is fixed by the oxalic acid. When the fluid which drops from the condenser has no longer an alkaline reaction, stop the process by removing the stopper from the flask. Care must be taken that at the end no alkali comes over from the flask containing the strongly alkaline fluid, as is rather apt to occur should there be bumping. Titrate the 100 cc. of decinormal oxalic acid with a decinormal soda solution, using methyl orange or neutral lacmoid as indicator. Every cc. of soda less than a hundred used represents ·0014 grm. of nitrogen.

Example.—Suppose that on titrating the oxalic acid solution with decinormal soda the neutral point is reached when 60 cc. of the latter have been added; the remaining 40 cc. of the decinormal oxalic taken must therefore have been neutralised by the ammonia derived from the nitrogen in the 5 cc. of urine, therefore the 5 cc. of urine contain ·0014 × 40 grms. nitrogen, or ·056 grm. If the total amount of urine passed in 24 hours be 1500 cc., this will contain 16·8 grms. of nitrogen.

About 15·20 grms. of nitrogen are excreted daily in the urine of a healthy adult on ordinary diet. A knowledge of the quantity excreted in disease is not of much value unless one has some idea of the amount of nitrogen in the diet. It must be remembered also that normally 1–2 grms. of nitrogen appear in the fæces.

Urea ($CO(NH_2)_2$).

Qualitative test for.—Place a drop or two of the suspected fluid on a slide and add one drop of nitric acid, warm gently. On evaporation rhombic or hexagonal crystals of nitrate of urea will be found if the latter body is present. (Fig. 89).

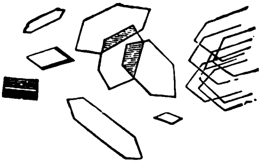

Fig. 89.—Urea nitrate.

Quantitative Estimation.

1. *From the specific gravity.*—An approximate estimation of the amount of urea may be made by dividing the last two figures of the specific gravity by 10—*e.g.* if the specific gravity of a given urine be 1020, it contains (approximately) 2% of urea; this only holds good in the absence of sugar, or much albumin, and if the patient is not very feverish.

2. *From the amount of nitrogen given off on treating the urine with hypobromite of soda*

This method depends upon the fact that urea is decomposed by hypobromite of soda according to the following equation—$CO(NH_2)_2 + 3NaBrO = 3NaBr + N^2 + 2H_2O \times CO_2$. It is found that under ordinary conditions 1 gramme of urea yields 371 cc. of nitrogen; all that is necessary, therefore, is to remove the CO_2 by means of an alkali (caustic soda), and to measure the remaining volume of gas and to calculate from it the amount of urea which was contained in the quantity of urine taken. The various forms of apparatus employed in carrying out the process differ chiefly in the method adopted for catching and measuring the nitrogen given off.

The number of cc. of nitrogen given off (from 5 cc. urine) multiplied by 0·056 = grms. of urea in

100 cc. urine, *i.e.* the percentage, and this multiplied by 4·375 = grains of urea in 1 oz. of urine.

The following are the chief forms of apparatus employed :—

(1) *Dupré's :* The apparatus consists of a cylinder filled with water, into which dips a burette open at the lower end, and connected above with a T-piece attached by one limb to a short rubber tube provided with a clip, and by the other connected with a small bottle closed with a rubber stopper. 25 cc. of hypobromite solution are measured into the bottle, and 5 cc. of urine into the little tube provided for it, and the latter is then lowered into the bottle and leant up against its side. The stopper is inserted in the bottle, and the level of the water, both in the cylinder and the burette, is then adjusted till it is opposite the zero mark on the burette. This is done by raising or lowering the burette, whilst the clip at the top is kept open. When the water is level the clip is closed, the urine and hypobromite mixed by inverting the bottle, and the gas collected. After a quarter of an hour, the burette is raised, till the water in it is again on a level with that in the cylinder, and the amount of gas evolved read off. The burette is graduated in lines, which show the percentage of urea, and by multiplying these by 4·375 one arrives at the number of grains per ounce.

(2) *Gerrard's ureometer* (Fig. 90).—This consists of a graduated glass cylinder closed at the upper end by a rubber stopper. Through the stopper there passes a T-tube. One limb of this tube is closed by a clip or stopcock, the other is connected to a piece of rubber tubing. The other end of the rubber tubing terminates in a piece of glass tube, which is inserted into the rubber stopper of a wide-mouthed flask of about 6 oz. capacity. From the lower end of the graduated cylinder another rubber tube passes to a short

wide glass tube open at its upper end. The object of this tube is to act as a reservoir of water. It can be slipped up and down upon the cylinder by means of a metal ring.

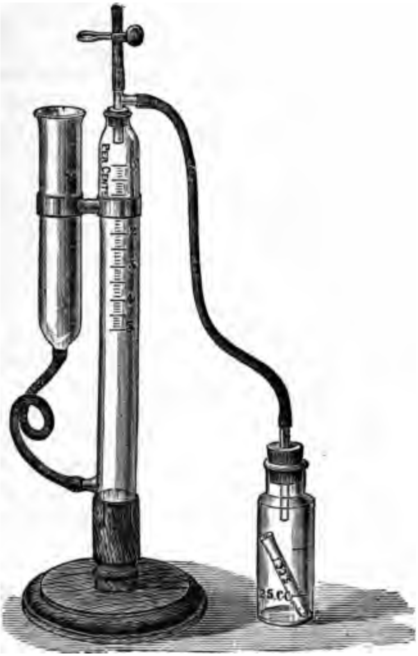

Fig. 90.—Gerrard's ureometer.

How to use the apparatus.—Place in the glass flask 25 cc. of hypobromite solution (Appendix, 12). An excess of hypobromite does no harm—one must merely be sure that enough is taken to decompose all the urea likely to be found in the urine. Measure 5 cc. of urine into the small glass tube provided for the purpose. If the urine contains albumin, the latter must be first removed. This is best done by taking a definite quantity of urine—say, 50 cc.—adding to it a drop or two of acetic acid, and boiling for a couple of minutes. Filter and make up to its original volume.

The small tube containing the urine must now be lowered into the flask. This is best done by inserting the point of the little finger—not too tightly—into the mouth of the tube. The latter must then be propped up against the inner surface of the flask so as to prevent the hypobromite solution from mixing with the urine. The reservoir of the graduated cylinder must now be filled with water. The stopper is then tightly inserted into the mouth of the flask. The clip or stopcock must now be opened, and the reservoir raised until the water inside the cylinder stands at the zero mark, and is level with that in the reservoir. The water must also be very low down in the latter, else there will be an overflow subsequently. Now close the stopcock and gently tilt the flask so as to allow the urine and hypobromite solution to mix. Great effervescence ensues, and the nitrogen liberated enters the cylinder and drives water out of it up into the reservoir. Wait for ten minutes to allow cooling to take place. Then lower the reservoir until the water in it and the cylinder are again level, and read off the amount of gas in the latter. The cylinder is graduated in percentage of urea. To get the number of grains per ounce, multiply this by 4·375.

In normal urine only 92 per cent. of the nitrogen of the urea is given off. If sugar be present, the yield is for some reason much larger, amounting to about 99 per cent. In Gerrard's instrument the scale is constructed for normal urine. In cases of diabetes, *therefore, it is* necessary to correct one's result by multiplying the figure obtained by $\frac{92}{99}$, *i.e.* by 0·93.

(3) *Ureometer of Doremus.**

This is a very simple and cheap form of apparatus, devised by Dr. Chas. Doremus, of New York. Modifications of it are sold by Southall (Birmingham) and Cooper (London). It consists of a bent tube with a long limb closed at its upper end, and a short limb which expands into a wide bulb with an open mouth.

In using the instrument it must first be filled with hypobromite solution. Hold the tube vertically, and pour in the solution just short of overflowing. Then gently incline the instrument so that the solution fills the long limb as far as the mark near the bend, and no air bubbles are present. A little water should then be added to fill the rest of the bend and the lower part of the bulb. The instrument may now be fixed in its stand.

One cc. of urine has next to be measured out with the pipette provided. This is where the difficulty in using the instrument comes in. In order to accomplish it successfully, proceed as follows:—Slip the rubber nipple high up upon the pipette. Compress the nipple, and immerse the point of the pipette just below the surface of the urine. Then allow the nipple to expand fully. Urine will be drawn up beyond the mark. Now slide the nipple bodily down with the finger and thumb over the rimmed end of the pipette. This displaces the urine. Continue the displacement till the mark is reached. The pipette will now keep itself charged with exactly 1 cc. of urine. Wipe the outer surface of the pipette, and insinuate the end of it as far as it will go into the long limb of the apparatus. Now compress the nipple so as to squeeze out all the urine, and then withdraw the pipette, keeping up the compression until it is out of the liquid.*

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* Also known as Southall's or Thursfield's Ureometer. A modification of this apparatus by Hinds has many advantages over *the original.*

In about a quarter of an hour the amount of nitrogen which has collected in the long limb may be read off. The instrument is provided with an English scale representing grains per ounce, and also with a metrical scale. The large divisions on the latter represent centigrams of urea per cc. of urine. The scale between is subdivided into tenths. Suppose the reading to be ·025. This means ·025 grm. of urea in 1 cc. of urine, and multiplying by 100, = 2·5 per cent. If more than 3 per cent. of urea is present, the urine should be diluted with an equal volume of water and the result multiplied by 2. Albumin, if present, should always be removed beforehand.

About 450 grains (25–40 grms.) of urea are excreted daily in health. This is about 2 per cent., or 9 grains per ounce. It is increased when much food or water is taken. Also in fevers, in diabetes, and in poisoning by phosphorus or arsenic. It diminishes under diminished diet, in some severe diseases of the liver (because less is formed), and in some conditions of the kidney. No calculation of the amount of urea which a patient is passing is of much use unless the amount of nitrogen in his diet is also taken into account.

Uric acid ($C_5H_4N_4O_3$) occurs in the urine in combination with alkalies. Being a dibasic acid, it forms two classes of salts, the normal urates ($Na_2\overline{U}$), and the acid urates ($NaH\overline{U}$). Under certain conditions uric acid becomes free in the urine, and separates out as a crystalline deposit ("cayenne-pepper" deposit). Acid urates may also separate out in a crystalline form. Both forms of separation, when occurring inside the urinary passages, lead to the disease known as "gravel" or to stone formation. The microscopic characters of these deposits will be described in another section. The conditions which favour the separation of uric acid and acid urates are (1) the presence of a large amount of uric acid in the urine; (2) a high degree of acidity; (3) the presence of little salts, and of a small amount of pigment.

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* The pipette may be provided with a piston instead of a nipple, or one can extemporise a piston by slipping over the end of the pipette a piece of thick-walled rubber tubing in which a piece of glass rod, lubricated with glycerine, can be made to act as a piston *and suck up the urine.*

Qualitative test for uric acid (murexide test).—Evaporate the suspected fluid to a small bulk. Place five drops of it in a porcelain basin, and add one drop of nitric acid. Evaporate very slowly, avoiding charring. When almost dry, add to the orange-coloured residue a small drop of ammonia. A purplish or rosy red colour appears at the edge of the drop, often better seen after gently heating.

The action which takes place consists in the oxidation of the uric acid into alloxantine ($C_8H_6N_4O_8$)—the orange-coloured residue. On adding ammonia, purpurate of ammonia is formed ($NH_4C_8H_4N_5O_6$), and produces the purplish red colour.

Quantitative estimation.—For clinical purposes this is best accomplished by the method proposed by Hopkins. It is based upon the insolubility of acid urate of ammonia in a saturated solution of ammonium chloride. By saturating a given quantity of urine with chloride of ammonium, all the uric acid separates out as acid urate of ammonium, which is collected; the uric acid is then split off from it, and estimated either by weighing or by titration with permanganate of potash. Proceed as follows:—

(1) Saturate 100 cc. of urine with powdered ammonium chloride. About 30 grms. will be required.

Saturation is complete whenever a few crystals remain undissolved after vigorous stirring at short intervals. Even if these should redissolve as the temperature of the mixture rises again after its initial depression, it does not matter.

(2) Render alkaline by adding a little ammonia.

(3) After it has stood for ten minutes, filter and wash precipitate several times with saturated solution of ammonium *sulphate.*

(4) Wash precipitate off filter with a jet of hot water, add a pinch of carbonate of soda, and heat till precipitate dissolves.

(5) Add distilled water to 100 cc.

(6) Add 20 cc. strong sulphuric acid.

(7) While hot, titrate with a $\frac{1}{20}$ normal solution of potass. permanganate—*i.e.* 1·578 grm. per litre.

(8) Stop whenever a pink colour lasting a few seconds has appeared. Subsequent disappearance of the colour is to be disregarded; it is sufficient that it should be visible for a second or two after stirring.

(9) Every cc. of the permanganate used$=$0·00375 grm. of uric acid.

The presence of bile pigment interferes with the titration with permanganate. In that case the uric acid must be estimated by weighing, and for the details of the method we must refer the reader to the original paper.* The trouble in the above method is the washing with ammonium sulphate. This must be continued till all ammonium chloride is removed, but the solution is so dense that it passes very slowly through the filter paper. To obviate this difficulty, Hopkins has described an abbreviation of the process which is accurate enough for clinical purposes. It is as follows:—

(1) Saturate 20 cc. of urine with chloride of ammonium, and add ammonia, as above.

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* *Journal of Pathology and Bacteriology*, June, 1893.

(2) Place a plug of glass wool in the neck of a small funnel, and wash it by filtering through it some saturated ammonium sulphate solution.

(3) After the saturated urine has stood for ten minutes, filter it through the glass wool. (A little experience is necessary to enable one to pack the glass wool firmly enough to keep back the precipitate, but yet not so tightly as to render filtration very slow.)

(4) Wash the precipitate with saturated sulphate of ammonium till no more chloride comes away.

(5) Transfer plug and precipitate bodily to a flask. Add 20 cc. water and a small pinch of carbonate of soda. Heat till the urate dissolves. Cool under the tap, and add 4 cc. of strong sulphuric acid.

(6) Titrate with $\frac{1}{50}$ normal permanganate solution, as above. (This is best made by diluting some of the strong solution from time to time.)

(7) Every cc. of the permanganate used = 0·0015 grm. uric acid.

The presence of albumin does not affect these methods. If there be a deposit of uric acid or urates in the sample of urine, the whole should be thoroughly shaken up, the amount to be operated with measured off, and saturated as usual. Or a few drops of ammonia may be added and the urine warmed till the deposit dissolves. A deposit of phosphates may be neglected.

From 0·4 to 0·7 grm. (7–10 grains) of urid acid is excreted daily. The amount is increased whenever a large destruction of nuclein is going on; thus in leucocythæmia as much as 4 grms. may be excreted daily. It is also increased in acute fevers. It is diminished in chronic gout and after the administration of quinine.

We would again warn the reader against the common error of assuming that a deposit of urates or uric acid necessarily indicates an increased excretion of the latter.

Xanthin bases.—Amongst the chief products of the disintegration of nuclein are uric acid and some basic bodies which may be termed the "nuclein" or xanthin bases. These substances differ from uric acid in being pretty strongly basic. Xanthin is the chief member of the group. It has the formula $C_5H_4N_4O_2$—*i.e.* one atom of oxygen less than in uric acid. The other members are hypoxanthin or sarkin ($C_5H_4N_4O$), guanin ($C_5H_5N_5O$), and adenin ($C_5H_5N_5 + 3H_2O$). These bases are sometimes spoken of along with uric acid under the term "alloxur bodies." These alloxur bodies contain between them from 1 to 5 per cent. of the total nitrogen in the urine. A method has been described for estimating them,* but it is not adapted for ordinary clinical use, nor has its accuracy been fully established. The results which it has yielded show that the amount of nitrogen eliminated in the form of uric acid is much more constant than that which appears in the form of xanthin bases. Usually a good deal more of the former is eliminated than of the latter, but the proportions vary greatly even in health, and may at times even be reversed. The xanthin bases are increased just as uric acid is in conditions associated with increased destruction of nuclein—*e.g.* in leucocythæmia. A milk diet causes the bases to increase while the uric acid diminishes. There is no constancy in their behaviour in gout.

The only other nitrogenous constituents of normal urine which call for mention are creatinin and hippuric acid.

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* *Ztsch. f. Phys. Chemie*, Bd. xx. Krüger-Wulff. *See also Neubauer & Vogel*, "Analyse des Harns," p. 828 *et seq.*, 10th Ed.

Creatinin ($C_4H_7N_3O$) is creatin minus water. It is one of the forms in which creatin is excreted, and most of it is derived from the creatin in flesh food. About 1 grm. of it is excreted daily. Variations are of no known clinical significance. It is of some importance as being one of the constituents of normal urine, which is able to reduce cupric oxide.

Hippuric acid ($C_9H_9NO_3$) occurs in the urine as hippurate of sodium. About ½ grm. of it is excreted daily. This amount is increased by the taking of benzoic acid as a drug, or of fruits—*e.g.* mulberries and cranberries — which contain aromatic acids.

4. Abnormal chemical constituents of urine.

I.—PROTEIDS.

Any or all of the proteids of blood plasma—serum albumin, serum globulin, and fibrinogen—may occur in the urine. In addition, one meets with the compound proteids—mucin and nucleo-albumin, and with albumoses, both primary and secondary. It is doubtful whether true peptone ever occurs in the urine. It is true that two of these—mucin and nucleo-albumin—are to be regarded as normal urinary constituents, being added to the renal secretion as it passes along the urinary passages, but in health they are present in such small amount that they may be neglected. It is also true that any of the proteids mentioned above may occasionally find their way into the urine even in perfectly healthy persons; the urine of healthy infants, for instance, often contains traces of albumin. Into the possible causes of this, and into the distinction between "functional" and "organic" albuminurias, we do not *propose to enter.* Chemical examination of the urine can merely show the presence in the urine of a proteid; it cannot tell us to what its presence is due.

(1) **Serum albumin and serum globulin in the urine.**—These proteids may be found in the urine either together or separately. The former condition is the usual one, and constitutes what is ordinarily spoken of as "albuminuria." The relative proportion of each proteid varies greatly in different cases, but usually serum albumin is present in larger amount than serum globulin. The variations in their relative amounts have no clinical significance. In what follows, the term "albumin" will be held to include also globulin, unless stated otherwise.

Separation of albumin and globulin.—If one wishes to do this, the best plan is to render the urine slightly alkaline with ammonia, filter off phosphates if necessary, and add to 100 cc. of the filtrate its own volume of a saturated neutral solution of ammonium sulphate. By the end of an hour globulin, mixed, perhaps, with some mucin bodies, has separated out. Collect it on a weighed, dry, ash-free filter, wash with half-saturated ammonium sulphate solution, dry, and weigh. Then incinerate, and deduct the weight of the ash. The difference gives the amount of globulin present. If the total proteids have been estimated by *complete* saturation of another sample of urine with ammonium sulphate and treatment of the precipitate in the above way, the difference between the total proteids and globulin may be reckoned as albumin.

A simpler, but for clinical purposes sufficiently exact, method is that of Brandberg. It is thus described by Carstairs Douglas*:—"Brandberg's method is based on the fact that in the cold nitric acid test the ring of albumin appears the quicker the more proteid there is present. It has been found experimentally that if 0·0033 per cent. of albumin is present the ring appears in two to three minutes. By making a series of dilutions of definite strength of an unknown urine an estimate may be made of the amount of albumin present in it. For example, if the urine shows the ring of coagulation when diluted ten times, it contains $0·0033 \times 10 = 0·033$ per cent. albumin; if when diluted 30-fold it shows the same, it must contain $0·0033 \times 30 = 0·1$ per cent., and so on. In estimating the amount present in any unknown specimen, it is necessary first of all to make certain dilutions of it, and Tappeiner recommends that, to begin with, dilutions of 10-fold, 30-fold, and say 150-fold, should be made. If now, after trying these specimens with the acid, it is found that the ring appears in less than two or three minutes in the former two, and in more than three minutes in the 150-fold dilution, the proper point will be reached at a dilution somewhere between 30-fold and 150-fold. We may find it, let us say, at a dilution of 80-fold; the percentage of albumin will then be $0·0033 \times 80 = 0·26$. In any case the amount of albumin per cent. is obtained by multiplying 0·0033 by the degree of dilution."

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* "*Chemical* and Microscopical Aids to Clinical Diagnosis" (*Maclehose and Sons*), 1899, p. 70.

In carrying out the above procedure certain precautions must be observed:—

(*a*) The dilution of the urine must be made very exactly by use of a burette.

(*b*) About three-quarters of an inch of nitric acid should be used in each test, and the diluted urine run on to its surface very cautiously.

(*c*) Holding a watch in one's hand, one must know the exact time required for the appearance of a faint *but distinct* ring, and this time must fall between two and three minutes. The test tube should be held *meantime against* a dark surface.

Having estimated the total proteid present by this method, one must saturate some of the fresh urine with magnesium sulphate, filter off the precipitated globulin, and make a fresh estimate by the same method in the filtrate. This gives the albumin alone, and the difference between this and the previous result indicates the amount of globulin. The presence of the magnesium sulphate does not seem materially to affect the result. (Neubauer & Vogel.)

Tests for albumin in the urine.—A great many reactions have been proposed for this purpose, and much has been written on the subject. The fact seems to be that while in the majority of cases the presence of albumin or globulin in the urine can be demonstrated with perfect certainty by the application of one or two simple tests, yet every now and then cases are met with in which some doubt exists. We believe that by carefully applying the four following tests the presence of albumin and globulin can be clearly shown even when present in very small amount. Before proceeding to apply any of the tests, it is a *sine quâ non* that the urine should be *absolutely clear.* It may therefore be necessary to filter it. If it be acid, this can be proceeded with at once; should it be alkaline, enough acetic acid should first be added to render it faintly acid. It may be necessary to filter more than once. Should the urine still not be clear, the turbidity is probably occasioned by the presence of bacteria. These can best be removed either by (1) simply shaking up the urine with powdered barium carbonate, and filtering, or (2) adding to the urine a little caustic soda till a precipitate of earthy phosphates appears. This carries down with it all bacterial *débris*, and on filtering, the urine will now be found to be clear. It should then be slightly acidified with acetic acid, and the following tests proceeded with:—

(i.) *Boil* an inch or so of the urine in a test tube If it remains perfectly clear while the reaction is still acid, no albumin is present. Turbidity may be due to coagulated albumin or to the throwing down of earthy phosphates. Add a drop of nitric acid. Any turbidity which remains is due to the presence of albumin; mucin and nucleo-albumin are re-dissolved by the nitric acid.

(ii.) *Heller's test.*—Place a quarter of an inch of pure nitric acid in a test tube. With the aid of a pipette allow some of the urine to flow on to its surface. If after standing for half a minute no opaque white ring appears at the junction of the two fluids the urine may be regarded as free from albumin, for the test is capable of revealing the presence of ·002 per cent. If a ring forms it may be due to albumin, nucleo-albumin, or primary albumoses. A diffuse haze at the upper part of the fluid may be due to mucin. In the case of albumoses the ring disappears on heating, and reappears on cooling. (The method of distinguishing nucleo-albumin and mucin will be considered later.) If the urine be very concentrated, nitrate of urea or acid urates may separate out, usually in the form of a cloud without sharp margins. This can at once be distinguished by the fact that previous dilution of the urine with twice its volume of normal salt solution prevents its appearance. The precipitate also disappears on heating, and shows small crystals on examination with the microscope. The presence of resinous bodies—*e.g.* balsams—in the urine may also cause the appearance of a white cloud, which, however, is at once dissolved on the addition to the urine of half its volume of alcohol (rectified spirit).

It will often be noticed that a brownish-red transparent ring appears at the junction of the nitric acid and the urine. It is due to the oxidation of the urinary indigogens and the production from them of pigments. It occurs, therefore, in urines which are rich in indigogens, and has, of course, nothing to do with albumin. Such urines will be more fully considered later.

Effervescence on the addition of nitric acid may be due to decomposition of urea with the liberation of CO_2 and nitrogen. This only occurs if the nitric acid used contains some nitrous acid also.

As a substitute for nitric acid in the above test, one can use Spiegler's solution. It consists of—

Perchloride of mercury...	... 4	parts.
Tartaric acid	... 2	„
Glycerine	... 10	„
Water	... 100	„

It gives a distinct white ring if as little albumin as 1 in 350,000 is present, and may reveal the presence of albumin even in normal urine.

(iii.) *Ferrocyanide of potash test.*—Take 2 in. of urine in a test tube. Add 10 drops of a 5 per cent. solution of ferrocyanide of potash, and then render strongly acid with acetic acid. If the urine remains clear, no albumin is present. Turbidity may be due to albumin, albumose, or nucleo-albumin. Albumose may be distinguished by the nitric acid test; nucleo-albumin by the fact that it is precipitated by acetic acid alone without the aid of ferrocyanide.

(iv.) *Picric acid test.*—(Use a saturated solution of pure picric acid, without the addition of citric acid, as in Esbach's solution.) Place 2 in. of the picric acid in a test tube. Allow the urine to drop into it from a pipette. If no cloud forms around the drops, the urine is albumin-free. A cloud may be due to albumin, albumoses, or peptone. The precipitate produced by the higher albumoses and peptone disappears on heating. Quinine also gives a precipitate with picric acid which disappears on heating.

It will be observed from what has been said above that it is comparatively easy to be sure of the absence of albumin—not so easy to be certain of its presence. The substance which is most apt to be mistaken for serum albumin in the urine is nucleo-albumin. We have already indicated some methods of distinguishing between the two, and shall return to the subject later when we consider "nucleo-albuminuria."

Quantitative estimation of albumin.—This can be done with sufficient accuracy for clinical purposes by means of Esbach's albuminimeter. The principle of the method consists in measuring the depth of the coagulum produced in the urine by the addition of picric acid. The instrument consists of a thick glass test tube, with graduations on it from 0 up to 7.

Method.—Filter the urine if not already clear, and if alkaline render slightly acid with acetic acid. If the specific gravity is 1010 or more, dilute the urine sufficiently to bring the density below that level (to 1008). This is important, and is often overlooked. Fill the tube with the urine up to the mark U. Pour in the reagent (Appendix, 13) up to the mark R. Close the tube with a rubber stopper, and gently invert it a few times to allow the fluids to mix. Set aside for twenty-four hours. At the end of that time read off the level of the surface of the precipitate. The figures on the scale represent grammes of dried albumin per litre of urine.

Divide by 10 to get the percentage, and multiply the result by 4·375 to get the amount of albumin in grains per ounce of urine. If the urine requires to be diluted, the result must, of course, be multiplied the requisite number of times.

Very small quantities of albumin cannot be estimated by *Esbach's* method, as the instrument does not record less than 0·1 per cent. If after the first trial the level of the precipitate is found to be above the mark 4, the urine must be diluted and a fresh estimation made.

An excretion of 8 grms. of albumin daily represents an ordinary degree of albuminuria. This is equivalent to about ½ per cent.

(2) **Albumosuria.**—This is a more correct term than "peptonuria," which was formerly in use. It is very doubtful whether true peptone ever occurs in the urine at all. The clinical significance of the presence of albumoses in the urine is not yet finally determined. Recent investigations tend to show that they may occur in any "infective" disease—*i.e.* wherever disintegration of tissue is going on under the action of micro-organisms. Thus they are not uncommonly met with in the urine in pneumonia. They are most constant, however, in cases where a large collection of pus has formed in the body—*e.g.* in empyema or large abscess formation. They have also been found in considerable quantity in some cases of nephritis. The albumose in these cases is usually transient and of little importance. Permanent and abundant albumosuria may almost be regarded as pathognomonic of diffuse sarcomatous degeneration of the bone-marrow (myelopathic, or Bence Jones', albumosuria).

Detection of albumoses.—There are two classes of albumoses—primary and secondary—the latter standing nearest to the peptones. From a clinical point of view the differentiation of the two is of no importance, but they differ somewhat in their chemical reactions, and this necessitates the application of different tests for their detection. We will assume first that the urine to be examined is free from albumin. Proceed as follows:—

(i.) Add to the urine (filtered and acidified if *necessary)* a few drops of a saturated solution of picric acid. Boil and filter whilst hot. If the filtrate becomes cloudy on cooling, albumose is present.

The presence of antipyrin, quinine, and certain resins in the urine is apt to give a similar reaction.

(ii.) Apply Heller's test as already described (p. 329). A white cloud which disappears on heating and reappears on cooling indicates the presence of primary albumoses. The cloud is situated towards the upper part of the tube—it does not form a sharp ring close to the nitric acid as is the case with albumin. The secondary albumoses do not give this reaction unless in the presence of an excess of salt.

(iii.) Add to the urine an equal volume of a saturated solution of common salt, and then drop in acetic acid so long as a cloud forms. If this disappears on heating and reappears on cooling, albumoses are present. Both forms of albumose give this reaction.

If the urine is already albuminous the albumin should be removed before testing for albumose. To do this bring the urine to boiling point, add a drop or two of acetic acid, and boil for two minutes. Filter and test filtrate as above. The small quantity of albumose likely to be formed from the albumin during the boiling does not vitiate the result. If we wish to avoid the possibility of such a fallacy, we can add to the urine its own volume of 10 per cent. trichloracetic acid, rapidly bring to the boil, and filter hot. Test the filtrate, after cooling, for albumoses.

The Bence Jones albumose already referred to is a hetero-albumose. It may be present in enormous amounts, and sometimes appears in the urine as a precipitate. It gives the ordinary albumose reactions, but is characterised by its behaviour on heating. Urine containing it becomes opaque at a comparatively low temperature (below 60° C.), and a sticky coagulum forms which floats on the surface, or adheres to the *sides of the* tube. If the reaction is acid this coagulum *disappears entirely or almost so on boiling, and reappears on cooling.* It is also very easily soluble in dilute alkali. The body also gives a sharp ring with strong hydrochloric acid, *even when the urine is diluted with twenty times its volume of water.* This ring disappears on heating and reappears on cooling (Bradshaw's reaction).*

If the presence of true peptone be suspected, the urine must be saturated while boiling with sulphate of ammonium and the filtrate tested for peptone by the ordinary reactions—the best being the occurrence of a white ring on floating the urine on Spiegler's solution; or, better, dialyse the urine for two hours and test the dialysate for peptone. No albumose passes through in that time. Very delicate reactions for the presence of albumoses and peptones in the urine have been described by Salkowski† and by Harris,‡ and may be referred to by those who are specially interested in the subject. We have had no personal experience of their use.

(3) **Nucleo-albuminuria and mucinuria.**—We have already mentioned that both a nucleo-albumin (or a substance very closely resembling one) and mucin occur normally in the urine, and it is probable that the so-called "mucus" of the urine consists mainly of the former. In catarrhal conditions of the urinary passages, however, and especially of the bladder, an excess of true mucus may appear in the urine, and to this the term "**mucinuria**" has been applied. As long as the urine is acid, mucin is insoluble and forms a deposit at the bottom of the vessel. Such a deposit may be distinguished from pus by the absence of pus cells on microscopical examination, and by the fact

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* *Trans. Royal Med. Chir. Soc.*, 1898 and 1899.

† *Centralb. f. d. Med. Wissenschaften*, No. 7, 1894.

‡ *American Journal of the Medical Sciences* May, 1896.

that on adding to it some caustic potash the solution is not decidedly ropy, as it is in the case of pus. If the urine is alkaline the mucin goes partially or entirely into solution. It may then be detected by adding to the urine a few drops of acetic acid. A white cloud, insoluble in excess and increased on boiling, indicates mucin. This often succeeds better if the urine be previously diluted with its own bulk of water, as the presence of a large quantity of salts tends to prevent the precipitation.* We have already said that **nucleo-albuminuria** is very apt to be mistaken for ordinary albuminuria. The reason of this is that nucleo-albumin gives most of the ordinary albumin reactions. Thus it gives a positive result with Heller's and with the ferrocyanide test. It may be distinguished by the fact that it is precipitated on the addition of acetic acid. It is rather more difficult to distinguish it from mucin. The latter is insoluble in excess of acetic acid, nucleo-albumin is soluble in large excess. This test, however, is not always quite satisfactory. Nucleo-albumin gives a sharp ring with Heller's test; mucin only a diffuse haze. Nucleo-albumin yields, as a rule, no reducing substance on boiling with mineral acid, and its ash contains phosphorus; mucin yields a reducing substance, and its ash is phosphorus-free. The significance of nucleo-albuminuria has not yet been fully made out. It seems to occur not unfrequently in febrile diseases or in conditions associated with a destruction of the secreting cells of the kidney.

"Fibrinuria" has already been described (p. 302).

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* Previous dilution of the urine has also the advantage that it prevents one from being deceived by a precipitate of urates, which may be thrown down if acetic acid is added to a concentrated urine.

II.—BLOOD AND ITS DERIVATIVES.

Blood may appear in the urine as a whole (hæmaturia), or blood pigment may appear without corpuscles (hæmoglobinuria). These two conditions can only be differentiated by examining the deposit for blood cells. There is here one source of fallacy. In alkaline urines, especially if they have stood for some time, the red cells are apt to swell up and disappear. The urine should, therefore, be examined as fresh as possible.

If a urine contains only a small amount of blood or blood pigment it has a peculiar opaque appearance, to which the term "smoky" is applied. Larger quantities of blood give to the urine a red colour varying in intensity with the amount of blood present. The blood corpuscles are apt to settle to the bottom, producing a flocculent deposit, which is brown or red according to the amount of the blood and the degree of its alteration.

The following tests depend upon the presence of blood pigment, and therefore give a positive reaction both in **hæmaturia** and in **hæmoglobinuria** :—

(1) *Heller's test.*—Place 2 inches of the urine in a test tube and render it strongly alkaline with caustic soda. Boil. If blood pigment is present the deposit is brownish-red in colour while the supernatant fluid is bottle-green.

The precipitate consists of earthy phosphates which have carried down with them hæmatin derived from the blood pigment, and are therefore reddish in colour instead of being white or yellowish. If the urine be alkaline a few drops of calcium chloride solution should be added to form more earthy phosphates. If the urine be very dark, as it may be (*e.g.* from the presence of bile), the supernatant fluid should be decanted off the precipitate and replaced by water. The test is sufficiently delicate to reveal the presence of 1 cc. of blood in 1 litre of urine.

Fallacies.—If the patient is taking senna, santonin, or rhubarb, the test may yield a positive result even although no blood is present. If the coloration is due to hæmoglobin, however, the precipitate yields the spectrum of alkaline hæmatin (Fig. 73), and this excludes all possibility of fallacy.

(2) *Guaiac test.*—Take 1 inch of urine in a test tube, add to it two drops of tincture of guaiac. A white precipitate forms, owing to partial precipitation of guaiac resin. Now add 1 inch of ozonic ether without shaking. If blood pigment is present a blue colour appears at the junction of the fluids.*

The blue colour is due to oxidation of the guaiac by oxygen derived from the ozonic ether, the blood pigment acting as the carrier. Ozonic ether is a solution of peroxide of hydrogen in sulphuric ether

Fallacies.—If iodides are present in the urine a blue colour is produced on applying the test. It is distinguished from that due to blood, (1) by the fact that it appears much more slowly, (2) by its appearing simultaneously all through the fluid, not at the junction of the ether and the urine.

Pus gives a greenish-blue colour with guaiac alone. It disappears on heating.

The presence of much saliva in the urine (*e.g.* from the patient spitting into it) is also a possible source of fallacy, as it gives the guaiac test.

Methæmoglobinuria.— Methæmoglobin may be formed from hæmoglobin in any acid urine after it has stood for some time. Not unfrequently, however, methæmoglobin is present in the urine when passed. It has been said to indicate that the hæmorrhage has its origin in the kidney. The characteristic smoky tint of the urine in hæmaturia of renal origin is largely due to methæmoglobin. The pigment present in "paroxysmal hæmoglobinuria" consists largely of methæmoglobin. Spectroscopic examination is the only satisfactory test for methæmoglobin. The urine if very dark in colour should first be diluted, and it should always be filtered. It should then be examined in a layer 5 cm. thick—a small flat glass bottle does well enough. If methæmoglobin is present there will be a band visible in the red, in addition to two bands nearly in the position of those due to oxyhæmoglobin (*see* Fig. 73).

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* The tincture of guaiac must be prepared from fresh—*i.e.* unoxidised—resin, and the ozonic ether must contain in solution peroxide of hydrogen of 30-volume strength. It should give off bubbles of gas when poured into the test-tube. If these points are not attended to the test may fail.

Hæmatoporphyrinuria.— Hæmatoporphyrin (iron-free hæmatin) occurs normally in the urine in very small amount, and may be considerably increased without affecting its colour. When present in large quantities the urine has a dark port-wine colour. Such a urine does not give the guaiac reaction. If examined with the spectroscope in a thin layer it may possibly show the characteristic spectrum of so-called alkaline hæmatoporphyrin, that being the form met with even in acid urines. Often, however, no distinct spectrum can be obtained on direct examination of the urine. In such a case the pigment can be extracted by shaking up the urine with a little amylic alcohol or acetic ether, after the addition of a few drops of acetic acid. The extract so obtained shows the bands of alkaline hæmatoporphyrin, viz.: 4 bands, one at the junction of the red and yellow, a second in the yellow, a third in the green, and a fourth (the broadest) between the green and the blue (*see* Fig. 73). On adding a drop or two of hydrochloric acid the bands of acid *hæmatoporphyrin* are obtained, viz.: 2 bands, one in the orange (narrow) and one at the junction of the yellow and green (broader). The latter is the characteristic band and consists really of two halves, a lighter half on the side next the narrow band and a very dark half on the side away from it.

Hæmatoporphyrin sometimes appears in large amount in the urine of patients who are taking sulphonal, but much more commonly in females than in males. It is a sign of very grave significance, as such cases often terminate fatally. The excretion of port-wine-coloured urine by a patient who is taking sulphonal is always an indication for the immediate stopping of the drug and for the free administration of alkalies.

Urine which contains blood or hæmoglobin contains also, of course, some albumin, and it is often difficult to say whether the blood is sufficient to account for all the albumin present or whether true albuminuria exists as well. We have found that if human blood be added to normal urine in an amount sufficient to produce distinct smokiness, the quantity of albumin amounts to merely a trace. Even when the quantity added is sufficient to render the urine distinctly red, the amount of albumin as shown by Esbach's method is only ½ per 1,000.

III.—Sugars in the Urine.

The only sugars which are of practical importance in the examination of the urine are glucose and lactose. It is possible that lævulose may sometimes occur along with glucose. Cane sugar and maltose may conceivably appear in the urine if excessive quantities of either be ingested. The occurrence has been described of the special varieties of sugar known as the *pentoses*, but these are only very rarely present, and are not yet of much clinical importance.

Glucose in the urine.—Glucose (dextrose or grape sugar), $C_6H_{12}O_6$, is by far the commonest variety of sugar met with in the urine. The condition is spoken of generally as "glycosuria." This must be distinguished from "diabetes." Diabetes—or, more correctly, diabetes mellitus—is a disease of which glycosuria is the chief symptom, but every patient with glycosuria has not necessarily got diabetes. It has long been disputed whether or not traces of glucose occur in normal urine. Recent researches appear to have finally settled the question. Traces of glucose *do* occur in normal urine, but not in an amount capable of detection by the reagents usually employed. If, therefore, glucose be detected by any of the tests we are about to describe, its presence may be regarded as pathological.

Tests for glucose in the urine.—A group of these tests depends upon the fact that glucose can become oxidised at the expense of certain metallic oxides, this oxidation occurring most easily at a temperature near boiling, and in the presence of free caustic alkali. Copper is the metal usually employed in the test. If one takes a solution of caustic soda and adds to it a few drops of a very dilute solution of sulphate of copper, a blue precipitate forms. This is hydrated cupric oxide ($CuO{\cdot}H_2O$). If now one boils the blue precipitate it becomes black from the separation of cupric oxide (CuO). In the presence of certain substances, however—such, for example, as a tartrate—the cupric hydrate formed on the addition of the sulphate goes into solution instead of being precipitated. A deep blue fluid then results, which remains unaltered on boiling. If an oxidisable substance such as glucose is present, however, the blue cupric hydrate is reduced on boiling to cuprous hydrate ($Cu_2O{\cdot}H_2O$), which is not capable of being *held in solution,* and accordingly appears as a yellow precipitate, or it may be further dehydrated to cuprous oxide (Cu_2O), which is red. Now, glucose is not merely capable of reducing cupric hydrate, but it is also one of the substances which, like the tartrates, is capable of holding it in solution. Hence, if a solution of glucose be rendered alkaline with caustic soda, and a few drops of cupric sulphate solution added, the cupric hydrate formed is dissolved and a blue solution results. On raising this to the boiling point the glucose reduces the cupric hydrate, and yellow cuprous hydrate or red cuprous oxide is precipitated.

Such is a brief account of the chemistry of the copper test for sugar. The two chief methods of applying it are :—

(1) *Trommer's test.*—Take 2 in. of the urine in a test tube, add $\frac{1}{8}$ of its volume of caustic potash, and then drop in carefully some 1 per cent. solution of sulphate of copper, shaking after each addition. If any considerable quantity of glucose is present the cupric hydrate formed is at once dissolved, and a blue solution results. Continue to add sulphate of copper until a little cupric hydrate remains undissolved. Boil the upper part of the fluid and it becomes yellow from separation of cuprous hydrate; and on prolonged boiling this becomes red (Cu_2O). If more cupric hydrate has been formed than the amount of sugar present is capable of holding in solution, the excess yields black cupric oxide on boiling, which obscures the result. Hence the advantage of Fehling's reagent in which solution of all the cupric hydrate is ensured by the addition of a tartrate. The formula for the reagent will be found in the Appendix (14).

If even a small quantity of glucose is present, the reduction in Trommer's test begins before the boiling point is reached. Glucose is the only substance likely to be found in the urine which will do this.

(2) *Fehling's test.*—As a preliminary to carrying out the test, one must always make sure that the reagent is good. This is necessitated by the fact that Fehling's solution alters on keeping, with the result that on boiling it deposits a precipitate of cuprous oxide. The exact nature of the alteration is not fully understood. To test the Fehling's solution add to it an equal volume of water, and boil for two minutes. If the solution remains clear, it is to be regarded as safe. Should a precipitate occur, a little more caustic soda should be added and the liquid filtered. It is then ready for use. Add to 1 in. of Fehling in a test tube a few drops of the urine (freed from albumin), and boil. If any considerable quantity of glucose is present, a yellow or red precipitate will appear. Should none be evident, add as much urine as there was Fehling, and boil for two minutes. Set aside. If after standing the solution still remains quite clear, there cannot be more than a mere trace of sugar present. "If the proportion of sugar," says Allen,* "be moderate—that is, under 0·8 per cent.—the precipitation of the yellow or red cuprous oxide does not take place immediately, but occurs as the liquid cools, the appearance being somewhat peculiar. The liquid first loses its transparency, and passes from a clear bluish-green to an opaque light greenish colour. This green milky appearance is said to be very characteristic of dextrose, but it would be more correct to say that its appearance indicated the presence of some substance interfering with the normal reaction of sugar." Uric acid, creatinin, and many other substances may act as such interfering agents. Their presence renders the indications of Fehling's test

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* "Chemistry of Urine," p. 61.

uncertain when only small quantities of sugar are present. Allen has accordingly proposed the following modification of Fehling's test by taking advantage of the fact that a slightly acid solution of cupric acetate will precipitate most of these "interfering" substances without affecting any form of sugar. He proceeds as follows :—

Heat 7–8 cc. of the urine to boiling in a test tube, and, without removing any precipitate of albumin, add 5 cc. of the cupric sulphate solution used in preparing Fehling. Partially cool the liquid and add 1–2 cc. of a saturated solution of sodium acetate, containing enough acetic acid to give it a feebly acid reaction. Filter. To the filtrate add 5 cc. of the alkaline tartrate mixture used for Fehling, and boil for twenty seconds. If more than 0·2 per cent. sugar is present, cuprous oxide separates before the boiling point is reached. With smaller quantities precipitation takes place during the cooling of the solution, which becomes greenish, opaque, and suddenly deposits cuprous oxide as a fine yellow precipitate.

Certain *precautions and fallacies* in the use of Fehling's test have still to be mentioned.

In the first place, the urine must be free from albumin. If necessary add a drop or two of acetic acid to the urine, boil and filter. Neutralise the filtrate with a little calcium carbonate.

Fehling's test cannot be applied to strongly ammoniacal urine, as the free ammonia would prevent precipitation of cuprous oxide. In that case, Pavy's modification should be employed (p. 348).

If the amount of glucose present be more than is required for reduction of all the cupric oxide, some of it is apt to be caramelised, especially on prolonged boiling. The whole liquid and precipitate then becomes of a dark brownish colour.

The fallacies attendant upon the use of Fehling's test are due to the fact that other substances in the urine besides glucose can reduce cupric oxide. The chief of these are uric acid, creatinin, and hippuric acid among the normal ingredients ; of the abnormal constituents, the chief are lactose, glycuronic and glycosuric acids, and the products of certain drugs—*e.g.* chloral, chloroform, glycerine, benzoic acid, salicylates, carbolic acid, etc.; "alkapton" urines also reduce Fehling. If the patient be taking no drugs, and if the urine be examined by Fehling's method as modified by Allen, the only substances likely to lead to error are glycuronic acid and lactose. If one is still in doubt, the following additional tests should be employed :—

(3) *Nylander's (or Böttger's) test.*—The reaction depends upon the power of glucose to reduce bismuth compounds in alkaline solution with the formation of the black suboxide. To 10 cc. of urine add 1 cc. of the reagent (Appendix 15), shake and boil for two or three minutes. If sugar is present, a fine black precipitate of bismuth suboxide settles down. This test is extremely delicate, and is not given by the reducing bodies often present in excess in normal urine. Glycuronic acid, lactose, and some drugs (*e.g.* rhubarb, senna, salol, turpentine, antipyrin, etc.), however, give a positive reaction.

(4) *Picric acid test.*—Take 1 in. of urine in a test tube. Add ¼ in. of saturated solution of picric acid and a few drops of caustic potash ; heat. If sugar is present, the solution becomes of a very dark red colour, owing to the reduction of the picric to picramic acid.

Fallacies.—(*a*) Normal urine causes some darkening of the fluid when the test is carried out as above. This is owing to the creatinin which it contains. The colour is not nearly so dark as that produced by sugar, and the solution always remains transparent when held up to the light. (*b*) If the picric acid is impure, it may darken spontaneously when heated with caustic potash. It is, therefore, well to test the picric acid employed before using it.

(5) *Phenyl hydrazin test.*—Place 60 cc. of urine in a 100 cc. breaker. Add 1 gramme of sodium acetate and rather less pure phenyl hydrazin hydrochloride (the colourless crystals are best). Place in a water bath, stirring from time to time, and scraping off any deposit which forms on the sides. Continue till bulk is reduced to 10 or 15 cc. Allow to cool, and examine deposit after two hours. If there are no crystals, sugar is absent.

The following is a simpler form of the test for clinical purposes:—

Dissolve ½ gramme of phenyl hydrazin hydrochloride and 1½ grms. of sodium acetate in a few cc. of water by aid of gentle heat. Add 5 cc. of the urine, and boil for three to five minutes. Set aside, and after the lapse of ten minutes crystals will have formed if sugar is present.

The crystals, which consist of phenyl glucosazon, are needle-shaped, and of a bright yellow colour. They tend to be arranged in stars or fans.

The test is extremely delicate, and will show the presence of even 0·01 per cent. of glucose. Glycuronic acid and pentoses also give crystals, but are rarely present. The crystals yielded by lactose are shorter, broader, pointed at both ends, and have less tendency to form stars.

Riegler * proposes the following modification of the test:—

In a small porcelain capsule place about 1½ grains of pure white phenyl hydrazin crystals along with 7 grains of sodium acetate. Add 20 drops of the urine, and heat to boiling. Place the capsule on a table, and add 20 or 30 drops of 10 per cent. caustic soda. If a red violet colour appears within a minute, sugar is present in pathological quantities.

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* *Deut. Med. Woch.*, No. 3, 1901.

(6) *Fermentation test.*—This is really the only absolutely certain test for glucose, that being the only fermentable substance which is ever found in the urine. Neither lactose nor glycuronic acid—the two substances most liable to be mistaken for glucose—is fermentable. The following precautions must be observed in carrying out the test:—(i.) The urine must be acid. Alkaline urine would putrefy; therefore render it acid, if necessary, by adding tartaric acid. (ii.) Boil the urine for ten minutes, so as to drive off any air it may contain. Use German yeast. Shake the urine up with a small piece of it, so as to form an emulsion free from lumps, then place the urine so prepared in a tube. Special fermentation tubes are manufactured. If one of these is not obtainable, an ordinary test tube inverted in a bath of mercury will do. A Doremus (Southall's) ureometer tube does extremely well. The long limb of it should be filled with the urine completely, no air bubbles being left. Set aside the tube in a warm place, and examine after a few hours. If a distinct bubble has appeared at the top of the tube, the urine is fermentable, and contains at least $\frac{1}{80}$ per cent. glucose. Care must be taken to ascertain that the yeast is active. It should be tested with a dilute solution of glucose. It is also well to have a control tube full of normal urine to which yeast has been added, as the yeast itself is apt to give off a little gas.

If these precautions are observed, the test is absolutely trustworthy and extremely delicate.

Quantitative estimation of sugar.

(1) *Fehling's method* consists in titrating the urine with a known quantity of Fehling's solution

at boiling temperature, and observing when all the cupric oxide has been reduced to cuprous oxide, as evidenced by the discharge of all the blue colour from the solution. As Fehling's is a standard solution, and as the exact quantity of glucose required for the complete reduction of a given quantity of it is definitely known, it is easy to estimate the amount of glucose present from the quantity of urine used up in the titration. One proceeds as follows:—

If ordinary diabetic urine is being examined, it should be diluted to the extent of 1 in 20 (5 cc. of urine to 95 cc. of water).* Fill a burette with it. Measure 10 cc. of Fehling's solution into a flask of 100 cc. capacity, add a little strong caustic soda solution,† and water to 50 cc. Boil. Whilst boiling, run in the urine till the fluid above the precipitate of cuprous oxide has lost all colour. Any return of colour on cooling must be disregarded. This gives one an approximate idea of the amount of urine required for reduction. The process must now be repeated on a series of flasks, the urine in each case being added all at once, not dropped in gradually, until one finds to $\frac{1}{10}$ of a cc. the exact amount required to decompose the 10 cc. of Fehling.

This is the only way of getting a really accurate result, and the method is, of course, laborious. By having several flasks of Fehling ready and heated, a good deal of time can be saved, but even then a precise result can hardly be obtained within half an hour.

Calculation: 10 cc. Fehling = 0·05 grm. glucose. Suppose 10 cc. of the diluted urine has been used, and 5,000 cc. to be the amount of urine passed in 24 hours; then—

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* The urine must be diluted to such an extent that not more than 5–10 cc. are required to reduce 10 cc. of Fehling.

† This facilitates the separation of the cuprous oxide.

$$\text{Sugar in 24 hours} = \frac{5{,}000 \times 0{\cdot}05}{10} = 25 \text{ grms}$$

but the urine was diluted 1 in 20,

$$\therefore \text{ sugar in 24 hours} = 25 \times 20 = 500 \text{ grms.}$$

To get the result in English measure, remember that— 10 cc. Fehling = 0·77 grain sugar,

28·395 = cc. in 1 oz.

Therefore—

$$\frac{\text{Grains sugar}}{\text{in 24 hours}} = \frac{\text{ounces of urine in 24 hours}}{\text{number of cc. used,}} \times 0{\cdot}77 \times 28{\cdot}3$$

$$\textit{i.e.} = \frac{\text{ounces in 24 hours}}{\text{number of cc. used}} \times 21{\cdot}8.$$

The result must be multiplied according to the degree of dilution.

(2) *Pavy's method* is much more convenient for clinical purposes. It differs from that of Fehling in this—that a quantity of strong ammonia is added to the alkaline cupric tartrate solution. Ammonia is able to dissolve cuprous oxide, the solution being colourless. Hence, when the solution is titrated at the boiling point with a solution containing glucose, the mixture gradually fades until every trace of blue has gone, and one is not confused by the throwing down of a red precipitate. Pavy's solution has also the advantage of keeping indefinitely, and of being possessed of an originally deeper colour. The method of preparing it will be found in the Appendix (15).

Method.—Fill a burette with diluted urine as before. The outflow from the burette should be regulated by a stopcock or screw. The end of the burette is connected with a tube passing through the stopper of a 150 cc. flask. Another hole in the stopper allows the passage of an exit tube for the escape of the fumes of ammonia (Fig. 91). Place in the flask 10 cc. of the solution, diluted with 20 cc.

water.* When the solution is boiling, run in the urine. It should be run in at the rate of 60–100 drops per minute. Not too fast, or the limit of reduction may be overstepped, and not too slowly, or all the ammonia may be driven off and some cuprous oxide thrown

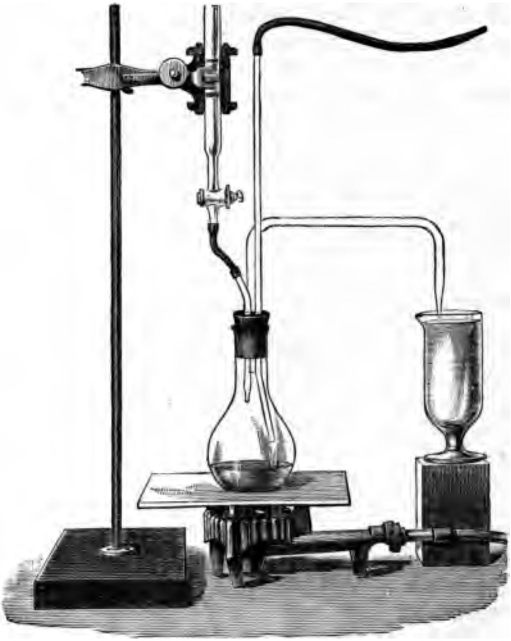

Fig. 91.—Pavy's Apparatus.

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* Some writers recommend that 50-100 cc. of Pavy's solution be taken. This necessitates the addition of a little talc or pumice stone to prevent bumping, and also of some arrangement to neutralise the fumes of ammonia given off. A jar of acidulated water (as shown in figure) does this quite well. A layer of

down. When the blue colour has entirely faded, take a reading of the burette.

The calculation is performed in the same way as for Fehling's method, but it must be remembered that 10 cc. of Pavy's solution is only = 0·005 grm. glucose, *i.e.* it is ten times less strong than Fehling's. The accompanying table saves the trouble of calculation. It gives the sugar in grammes per 1,000 cc. The same table may be used for Fehling's method (provided that 10 cc. of Fehling has been taken), the number of parts per 1,000 being always multiplied by 10, to allow for the greater strength of Fehling's solution; *e.g.* if 10 cc. diluted urine is used in Pavy's method, reference to the table shows that this means 0·5 grm. sugar per 1,000 cc.; by Fehling's method, it would be equivalent to 5 grm. per 1,000 cc. In each case the result must be multiplied by 20 if the urine has been diluted 1 in 20, and if it be desired to express the result in grains per ounce the amount per 1,000 cc. should be multiplied by 0·4375.

In both methods the urine must be freed from albumin (if necessary) by adding two drops of acetic acid, boiling, neutralising with calcium carbonate, filtering and making up to the original volume with water.

(3) *Gerrard's cyano-cupric method.**—This method depends upon the fact that the colourless double cyanide of potash and copper is capable of holding cuprous oxide in solution just as ammonia does

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liquid paraffin poured on its surface serves as a valve and prevents any air from getting in and causing re-oxidation of the cuprous oxide. Sutton leads the fumes away by an elastic tube closed at the end by a plug of glass rod, with a transverse slit just above the plug. This allows the fumes to escape, and prevents the return of fluid in case of a vacuum forming. Pavy himself uses only 5-10 cc. of his solution. If the urine is sufficiently diluted this gives quite accurate results, and the small amount of ammonia given off is no inconvenience.

* *See* Allen's "Chemistry of Urine," p. 74.

Table showing the Amount of Sugar expressed in Parts (by weight) per 1,000 *(by volume), corresponding with cc., in* 10*ths, required to decolorise* 10 *cc. of the Ammoniated Cupric Test.*

cc. to decolorise.	Parts per 1,000.	cc. to decolorise.	Parts per 1,000.	cc. to decolorise.	Parts per 1,000.
1·0	5·000	5·7	·877	10·4	·480
·1	4·545	·8	·862	·5	·476
·2	4·166	·9	·847	·6	·471
·3	3·846	6·0	·833	·7	·467
·4	3·571	·1	·819	·8	·462
·5	3·333	·2	·806	·9	·458
·6	3·125	·3	·793	11·0	·454
·7	2·941	·4	·781	·1	·450
·8	2·777	·5	·769	·2	·446
·9	2·632	·6	·757	·3	·442
2·0	2·500	·7	·746	·4	·438
·1	2·380	·8	·735	·5	·434
·2	2·272	·9	·724	·6	·431
·3	2·173	7·0	·714	·7	·427
·4	2·083	·1	·704	·8	·423
·5	2·000	·2	·604	·9	·420
·6	1·923	·3	·684	12·0	·416
·7	1·851	·4	·675	·1	·413
·8	1·785	·5	·666	·2	·409
·9	1·724	·6	·657	·3	·406
3·0	1·666	·7	·649	·4	·403
·1	1 612	·8	·640	·5	·400
·2	1·562	·9	·632	·6	·396
·3	1·515	8·0	·625	·7	·393
·4	1·470	·1	·617	·8	·390
·5	1·403	·2	·609	·9	·387
·6	1·388	·3	·602	13·0	·384
·7	1·351	·4	·595	·1	·381
·8	1·316	·5	·588	·2	·378
·9	1·281	·6	·581	·3	·375
4·0	1·250	·7	·574	·4	·373
·1	1·219	·8	·568	·5	·370
·2	1·190	·9	·561	·6	·367
·3	1·162	9·0	·555	·7	·364
·4	1·136	·1	·549	·8	·362
·5	1·111	·2	·543	·9	·359
·6	1·086	·3	·537	14·0	·357
·7	1·063	·4	·531	·1	·354
·8	1·041	·5	·526	·2	·352
·9	1·020	·6	·520	·3	·349
5·0	1·000	·7	·515	·4	·347
·1	·980	·8	·510	·5	·344
·2	·961	·9	·505	·6	·342
·3	·943	10·0	·500	·7	·340
·4	·925	·1	·495	·8	·337
·5	·909	·2	·490	·9	·335
·6	·892	·3	·485	15·0	·333

in Pavy's process. If, therefore, Fehling's solution is titrated with a sugar solution in the presence of this cyanide, the blue colour fades gradually, no precipitate being thrown down. The end point is thus very sharp, and as there is no tendency to reoxidation, the process may be safely conducted in an open porcelain basin.

To prepare the double cyanide.—Dilute 100 cc. Fehling with about 300 cc. water, boil and run in cautiously an approximately 5 per cent. solution of cyanide of potash. When the colour is just gone, dilute to exactly 500 cc. and keep in a well-stoppered bottle.

Method of titration.—To 50 cc. of the above solution add 10 cc. of Fehling, and boil in a porcelain basin. Cautiously run in the diluted urine till the colour of the solution has just gone, and calculate as in the ordinary Fehling method, for as the double cyanide undergoes no reduction, one has only to reckon with the 10 cc. of Fehling added. This process is both rapid and accurate, and will probably supersede all others for clinical use. As a mixture of the double cyanide and Fehling's solution will keep quite well for a number of weeks, it is easy to prepare it in bulk and to measure out each time such an amount as will correspond to 10 cc. of Fehling.

In an ordinary case of diabetes, 3 litres or so (about 5½ pints) of urine will be passed daily, containing on an average 100 grms. of glucose (3–4 per cent.).

In recording the amount of sugar passed in a case of diabetes it is better to make a really accurate estimation every third day, or even once a week, rather than a hurried and unreliable calculation every day.

Lactosuria.—Lactose is sometimes found in *appreciable* quantity in the urine of women who are nursing. It reduces Fehling's solution, and gives with the phenyl hydrazin test yellow crystals of phenyl lactosazon, which are broader than those yielded by glucose, but it gives no reaction with the fermentation test. It may be estimated by titration with Fehling's solution, it being remembered that the reducing power of lactose is to that of glucose as 7 is to 10; *i.e.* if 7 parts of glucose reduce a given quantity of Fehling, it will require 10 of lactose to effect the same result.

Pentosuria.—This is a condition only recently described. It consists in the presence in the urine of pentoses, *i.e.* carbohydrates containing only 5 atoms of carbon. They have the general formula, $C_5H_{10}O_5$. The pentoses do not exist free in nature, but can be obtained easily by hydrolytic decomposition of complex carbohydrates belonging to the gum class, present in many fruits (*e.g.* cherries). They are distinguished from the hexoses, such as glucose, in not furnishing lævulinic acid in decomposition with sulphuric acid or HCl; when gently heated with phloroglucin and HCl they give a cherry red reaction.

They furnish osazones, are not fermentable, and, on distillation with HCl, furfurol is given off.

They are optically active, and reduce cupric oxide. They only occur very rarely in the urine, and their pathological significance is unknown.

IV.—BILE IN THE URINE.

Both bile pigment and bile acids may be present Usually they occur together, but the pigment much more abundantly than the acids. The usual cause of the entrance of the bile constituents into the urine is some obstruction in the bile-passages. It was formerly believed that bile pigment could be formed in the blood owing to a destruction of the normal blood pigment, and thence find its way into the urine, constituting the so-called "hæmatogenous" jaundice. This is now known to be an error. It is also very doubtful whether traces of the bile acids are really always present in the urine, as is the belief of some. As long as the urine is fresh, bilirubin is the form of bile pigment always found in it. After it has stood for some time, biliverdin is apt to be formed as the result of oxidation.

Urine which contains bile is greenish or brownish-yellow in colour, and somewhat more viscid than normal, so that the froth which forms on the top after shaking is unusually permanent. Salol urine may closely resemble urine which contains bile, but the froth in the latter case is also greenish; in salol urine it is not.

Tests for bile pigment.

Gmelin's test.—Place some of the urine in a conical glass, and run a little impure nitric acid down the side so as to form a layer at the bottom. Oxidation of the bile pigment occurs, the most highly oxidised product (choletelin) forming a yellowish red ring nearest the acid. Above this is a reddish ring, then violet (bilicyanin), and highest of all, green (biliverdin). Of these rings the green is alone characteristic of bile, all the others may be yielded by urinary indigogens. The test, as thus carried out, is not very sensitive, and may fail even when 5 per cent. of bile is present. The sensitiveness of the reaction can be increased by repeatedly filtering the urine through an ordinary filter paper. The latter becomes impregnated with the bile pigment, and if a drop of impure nitric acid be placed upon it a play of colours can easily be seen.

The following modifications of it are much more delicate, and should always be employed in doubtful cases. They will reveal the presence of 0·2 per cent. *of bile.*

(1) To 50 cc. urine add 5 cc. of 10 per cent. barium chloride solution and 5 cc. chloroform. Shake for several minutes. Set aside for ten minutes. The chloroform and precipitate of phosphates fall down, carrying with them any bile pigment. If there is still any of the precipitate suspended, move the jar gently to and fro for a little, when it will settle down. Now draw off the chloroform and precipitate with a pipette; if some urine is removed at the same time, no matter. Place in a flat dish, and set the latter over a basin of hot water till all the chloroform has evaporated. Allow to cool, and pour off any fluid from the precipitate. The latter will be yellowish. Place impure nitric acid in drops here and there on the surface of the precipitate. If bile pigment is present, a play of colours appears round each drop.

(2) Render 10 cc. of the urine alkaline with caustic soda, and add a little 10 per cent. calcium chloride solution. Collect and wash the precipitate, and place it along with the filter paper in a small porcelain dish. Pour on it 10 cc. of acid alcohol (5 cc. strong soda to 100 cc. alcohol). Heat the yellowish solution in a test tube. If bile is present it becomes green or bluish.

Iodine test.—If a 10 per cent. alcoholic solution of iodine is poured on the top of the urine in a test tube an emerald green layer appears where the two fluids join, if bile is present.

Test for bile acids.

Pettenkofer's test for bile acids is inapplicable in the case of urine, for even normal urine gives, with strong sulphuric acid, a purplish colour which might be mistaken for a positive reaction. Oliver has proposed the following test as a substitute. It depends upon the power of bile acids to precipitate peptone in acid solution. The peptone solution is prepared as in the *Appendix* (16). Proceed as follows:—

Oliver's test.—Filter the urine until quite clear, acidify it if necessary, and dilute it till the specific gravity is less than 1,008. Take 60 minims of the solution in a test tube and add to it 20 minims of the urine. If bile acids are present a decided milkiness appears at once, and is dense in proportion to the amount of acids. It may disappear on agitation, but reappears on adding more of the solution. The test is extremely delicate, and nothing as yet found in the urine interferes with it.

V.—PUS IN THE URINE (PYURIA).

The naked-eye characters of a urine which contains pus have already been described (p. 305). On chemical examination such a urine is, of course, always albuminous. It is often difficult to decide, just as it is in hæmaturia, whether all the albumin is accounted for by the pus alone or whether there is true albuminuria in addition. Reinecke has proposed the following method for enabling one to form a conclusion in this matter. He shakes up the urine of twenty-four hours thoroughly, so as to diffuse the pus evenly through it. He then counts the pus cells present by means of a hæmocytometer, just as in the method for estimating the red blood corpuscles in the blood, only without previous dilution. He finds that 100,000 pus cells per cubic millimetre should correspond to 1 per cent. of albumin (Esbach). If there be more albumin than this with that number of corpuscles, then albuminuria is present in addition to pyuria. Obviously, the method can only afford approximate indications. Moreover, it is inapplicable if the urine be ammoniacal, or if it contains much mucus. It should be added that if the number of pus cells exceeds 3,000 per cubic millimetre, the urine should be diluted with 1 per cent. salt solution prior to counting.

Tests for pus.—As already mentioned, urines which contain pus give a green colour on the addition of guaiac, which, however, disappears upon heating.

If liquor potassæ be added to the deposit of pus, a ropy, gelatinous mass results. For the microscopical recognition of pus in the urine, *see* p. 373.

SOME RARER ABNORMAL CONSTITUENTS OF URINE.

(1.) **Urinary indigogens.**—We have seen (p. 311) that indol is excreted in the urine in the form of potassium indoxyl sulphate—the so-called "indican." Small quantities of potassium skatoxyl sulphate, derived from skatol, are also to be found in human urine. On oxidation these compounds yield coloured substances, indigo blue and indigo red. Hence they are called urinary indigogens. To detect their presence one oxidises them in one of these ways :—

(i.) Remove albumin, if present, by boiling. Add to some of the urine in a test tube an equal quantity of hydrochloric and a few drops of nitric acid, and boil. Cool and shake up with a little chloroform. The chloroform dissolves out the products of oxidation and becomes of a violet tint from the mixture of indigo blue and indigo red, if excess of indigogens is present. The presence of iodides in the urine must be excluded before applying this test.

(ii.) Remove albumin and add hydrochloric acid, as above. Then drop in slowly a *freshly prepared* dilute solution of bleaching powder (1 in 20), shaking all the time till the blue colour ceases to become deeper. The indigo blue may then be dissolved out with chloroform as above. The development of the blue colour does not proceed very rapidly, and one must be careful not to add too much bleaching powder, or oxidation will proceed too far, colourless compounds resulting.

Traces of the indigogens are normally present in the urine. The reddish-yellow transparent ring which appears above a layer of nitric acid when the latter is added to the urine is due to their partial oxidation.* They are increased in all conditions associated with excessive putrefaction. Hence they are much increased whenever the intestinal contents are unduly retained—*e.g.* in chronic constipation and intestinal obstruction. They are also increased in some fevers.

(2) **Acetone.**—Hydroxybutyric acid, aceto-acetic acid, and acetone may all occur in the urine. The relationship between the three may be seen from the following formulæ:—

β-Hydroxybutyric acid $= CH_3\,CH\,(OH)\,CH_2COOH.$
Aceto-acetic acid $= CH_3\,CO,\,CH_2COOH.$
Acetone ... $= CH_3,\,CO,\,CH_3.$

β-Hydroxybutyric acid is formed first, probably by destruction of proteids; it then becomes oxidised, yielding aceto-acetic acid.

$$CH_3CH(OH)CH_2COOH + O = CH_3COCH_2COOH + H_2O.$$

The aceto-acetic acid is very easily decomposed into acetone and CO_2

$$CH_3COCH_2COOH = CH_3COCH_3 + CO_2$$

Only aceto-acetic acid and acetone require to be detected in the urine. Hydroxybutyric acid always occurs along with the first of these.

Test for aceto-acetic acid.—The urine must be fresh and unboiled, as the acid readily decomposes. Take some urine in a test tube, drop in a solution of perchloride of iron, diluted until it is of a pale sherry colour, as long as a precipitate of phosphate of iron falls. Filter, and add to the filtrate another drop or two of the iron solution. The solution becomes claret coloured if aceto-acetic acid is present. On boiling the urine the colour disappears. Antipyrin, salicylates, carbolic acid, and some other drugs give a similar colour with perchloride of iron, but it is not affected by heat.

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* In part also the reddish colour so obtained is due to the pigment urorosein, which is produced from its chromogen by the action of mineral acids. Its clinical significance is unknown, but it is often met with in the urine of chlorotic patients and is probably of intestinal origin.

Test for acetone.—Urine containing acetone has a peculiar fruity odour. It reduces Fehling's solution. The best test for its presence is based upon its ready conversion into iodoform :—

$$C_3H_6O + 4KOH + 3I_2 = 3KI + KC_2H_3O_2 + CHI_3 \text{ (Iodoform)} + 3\,H_2O.$$

To 1 in. of the urine add five drops of 10 per cent. caustic soda or potash. Heat gently. Then drop in a saturated solution of iodine in potassium iodide until the liquid has a yellowish brown colour. Then add a little more caustic potash or soda. Iodoform appears as a yellowish turbidity, which settles down into a crystalline precipitate. It may be recognised by its odour. Under the microscope it consists of hexagonal plates often gathered into stars.

If only traces of acetone are present, it is better to distil the urine after the addition of a little phosphoric acid and test the distillate as above.

Acetone and the substances from which it is derived are especially apt to appear in the urine in cases of diabetes, and are to be regarded as of grave import, their appearance being often followed or accompanied by the development of coma. They are also found in some fevers.

(3) **Glycuronic acid.**—($C_6H_{10}O_7$) is probably derived in the body from dextrose. Mere traces of it exist in combination in normal urine. It is very prone to form ethereal or glucosidal compounds if suitable substances are introduced into the circulation. Hence

it appears in the urine in considerable quantity, in paired combination with aromatic substances, etc., after the administration of such drugs as chloral, benzoic acid, chloroform, morphia, etc. This circumstance gave rise to the old belief that such drugs produce glycosuria; in reality the substance which is excreted after their use is glycuronic acid, not glucose.

Occasionally glycuronic acid occurs spontaneously in the urine. It is then very apt to be mistaken for glucose. The error is a serious one, for the pathological significance of glycuronic acid in the urine is much less grave than that of glucose. Glycuronic acid reduces Fehling's solution, and gives a yellow crystalline precipitate with the phenyl hydrazin test. It can be best distinguished from glucose in the following ways :—

(i.) It does not ferment with yeast.

(ii.) Dissolve some *pure* phloroglucin by the aid of heat in 5 cc. of fuming hydrochloric acid so that a slight excess of the substance remains undissolved. Cool and divide into two equal portions. To one add ½ cc. of normal urine; to the other a similar quantity of the urine under examination after decolorising both with animal charcoal. Place both in a beaker of boiling water. In a few minutes the urine which contains glycuronic acid will show a red scum from which a bright red colour spreads throughout it. The normal urine remains unaltered.

(4.) **Cystin** $(C_3H_6NSO_2)_2$ is sometimes found as a deposit in acid urines. It is recognised by its characteristic crystals (*see* Fig. 96, p. 367). It is soluble in alkalies; hence the deposit disappears when the urine putrefies, an odour of sulphuretted hydrogen being evolved. If urine which contains it is boiled with a little caustic potash and acetate of lead, a *black precipitate* of sulphide of lead appears.

Cystin is a product of proteid metabolism, and is probably derived from cystein, the normal oxidation of which has somehow been interfered with. It is often associated with the appearance in the urine of diamines, such as cadaverin and putrescin. It is about twice as common in males as in females, and has rarely been observed after fifty years of age. It is apt to occur in several members of the same family, and may appear either persistently or intermittently. It is of little pathological significance except from its tendency to form calculi.

VI.—EHRLICH'S DIAZO REACTION IN URINE.

We shall first describe this test, and then state its significance. The reaction depends upon the fact that if sulphanilic acid (amido-sulpho-benzol) be acted upon by nitrous acid, diazo-sulpho-benzol is formed, which unites with certain aromatic compounds occasionally present in the urine to form aniline colours. Two solutions are necessary: (A) A saturated solution of sulphanilic acid in 5 per cent. hydrochloric acid; (B) a ½ per cent. solution of sodium nitrite. Both solutions should be as fresh as possible.

Add to some urine in a test tube an equal quantity of A; then add three drops of B, and shake till a froth forms. Render alkaline with ammonia. If the liquid becomes of a port wine colour while the froth is also red, the reaction is positive. The test has the following significance:—

(1) If the urine of a supposed typhoid in the second or third week fails to give the reaction, the diagnosis is probably wrong. In very mild cases, however, the reaction may be absent.

(2) The reaction is present in measles, but not

usually in German measles (rötheln). It is thus of value in distinguishing between the two.*

(3) It is very constantly present in tubercular disease which is advancing rapidly.

VII.—Drugs in the Urine.

Antipyrin.—After its use the urine may be red and dichroic, leading to the suspicion that blood is present. On adding a little dilute perchloride of iron a purplish red colour develops, which persists on boiling, but disappears on adding an acid. Urines containing antipyrin produce a partial reduction of Fehling's solution on boiling.

Carbolic acid (*see also* section on Colour of Urine, p. 298).—The best test for it is to add a little bromine water. The appearance of a whitish precipitate (tribromophenol) indicates the presence of phenol.

Chloral, chloroform, etc., lead to the appearance of glycuronic acid (p. 360).

Bromides.—Add a little hydrochloric acid and a few drops of a weak solution of bleaching powder. Shake with chloroform, and the latter becomes brownish red from the solution of the free bromine.

Iodides.—Acidify the urine with a little pure nitric acid, and shake up with chloroform. The latter becomes of a rose-red colour.

Iron.—Add a few drops of nitric acid. Boil, cool, and add a little 10 per cent. ferrocyanide of potash. A precipitate of Prussian blue forms if iron is present.

Rhubarb and santonin have been referred to under the colour of the urine (p. 299).

Salicylates and **salol** appear in the urine as salicyluric acid. Such a urine gives a bluish violet colour on the addition of a little perchloride of iron; it also partially reduces Fehling's solution.

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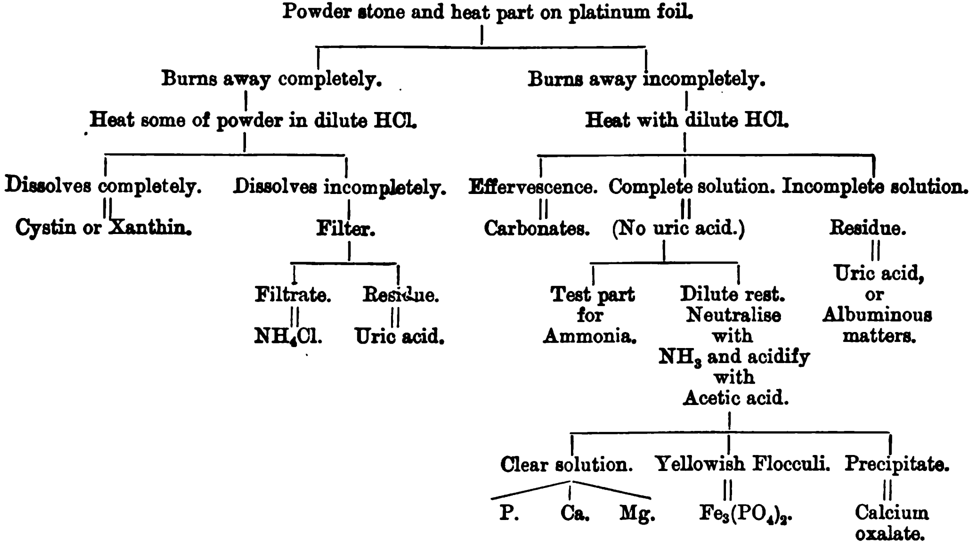
* Dr. Lloyd Jones informs us that he has found the reaction *in not a few cases of* German measles.

SCHEME FOR ANALYSIS OF URINARY CALCULI. (*After Salkowski.*)



Tannin gives a bluish black colour with perchloride of iron.

SECTION III.—MICROSCOPICAL EXAMINATION OF URINARY DEPOSITS.

The examination of deposits in urine and other fluids liable to decomposition is greatly facilitated by using a centrifuge. For ordinary clinical purposes, perhaps Beck's is as convenient as any; where a large number of examinations are undertaken it is better to employ one driven by electricity or water power.

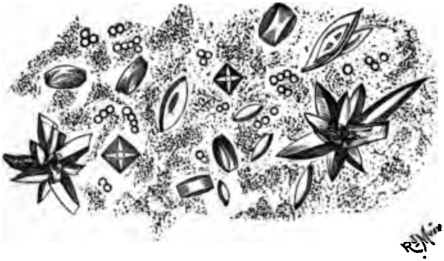


Fig. 92.—Deposit in acid urine.

1.—**Unorganised Deposits.**—The first group of urinary deposits includes the various salts and crystalline substances that are found in urine, either when freshly voided, or more often when it has stood for some time. The following occur in acid urine (Fig. 92).

(1) **Uric Acid.**—This appears under a variety of forms, and, unless the urine is almost devoid of colouring matter, assumes a reddish-brown colour in consequence of its absorbing a considerable amount of pigment. To the naked eye the appearance *resembles* that of a shower of grains of cayenne

pepper collected at the bottom of the specimen. Under the microscope the crystals are either rhombic prisms or some modification of that form. Often the more obtuse angles are rounded off and the edges continued in curved lines, so that pointed oval shapes result. Numerous crystals may be joined together to produce rosettes and other composite forms. Some of the more common are represented in the accompanying figure (Fig. 93).

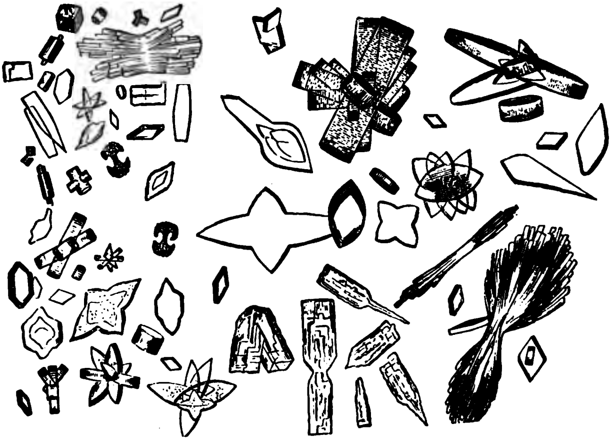


Fig. 98.—Uric cid. (*Finlayson after Funke.*)

(2) **Urate of soda** occurs rather frequently in the urine of recently born infants, when it produces a yellow stain on the napkin. In adults it is found very seldom. The appearance presented under the microscope is that of spheres, either solitary or in clusters, having a more or less crystalline structure, and possessing numerous spines radiating from their surface (*Fig. 94*).

(3) **Amorphous urates.**—These are urates of potassium, sodium, and ammonium. They have a considerable affinity for the urinary pigments, and hence are generally more or less pink or brick-coloured. In very pale urines they are colourless, and resemble rather closely a deposit of phosphates. Microscopically they consist of small granular particles, arranged in moss-like clumps. On heating a urine from which they have separated out, they will be found to redissolve before the boiling point has been reached.

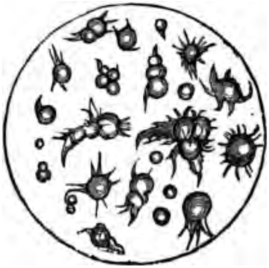


Fig. 94.—Urate of soda. (*After Roberts.*)

Uric acid and urate of soda can be preserved in Canada balsam — the water being got rid of by passing them through alcohol, then letting a drop dry on the slide, and adding balsam in xylol.

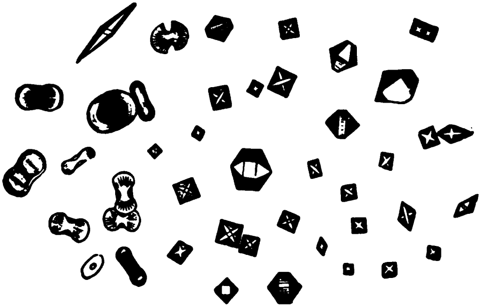


Fig. 96.—Oxalate of lime. (*After Finlayson.*

(4) **Hippuric acid** appears in human urine *chiefly after* the administration of benzoic acid or its

salts. It occurs as colourless four-sided prisms, insoluble in hydrochloric acid, but soluble in ammonia.

(5) **Oxalate of lime.**—This deposit is rarely abundant. The small colourless crystals lying on the top of the mucous deposit that settles at the bottom of the urine glass give the impression of an undulating snowy surface. They also adhere to irregularities on the surface of the glass, producing the appearance of scratches. Two forms are found under the microscope. The first, which is by far the commoner, consists of small octahedral crystals. When, as is generally the case, they are slightly flattened along one axis, they appear like squares crossed by two diagonal lines, or like long octahedra, according as the short axis lies in or perpendicular to the line of sight. The other form in which oxalates occur is that of minute dumb-bells or oval biscuit-shaped discs. Some writers consider that this form is not really due to oxalate, but to carbonate of lime; yet, though carbonates frequently enough assume this shape, there can be little doubt that under certain

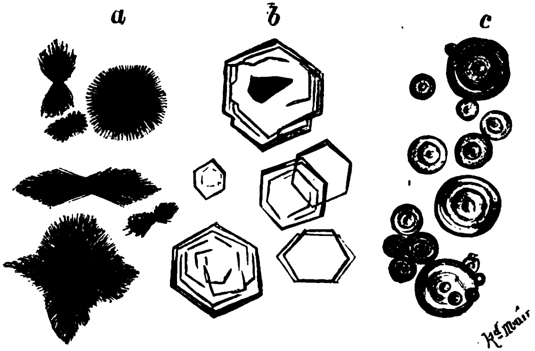


Fig. 96.—*a*, Tyrosin crystals; *b*, cystin; *c*, leucin.

conditions oxalates do so too. For permanent specimens octahedral oxalates are best mounted in glycerine jelly, dumb-bell oxalates in balsam (Fig. 95).

(6) **Cystin** is a rare deposit in human urine, but when it occurs the precipitate is often copious, and is not unlike a sediment of fawn-coloured urates. The addition of a few drops of acetic acid to a urine containing cystin in solution determines its precipitation. From urine it is deposited as hexagonal tablets, soluble in ammonia, and re-crystallising when the ammonia evaporates as hexagons or prisms (Fig. 96, *b*).

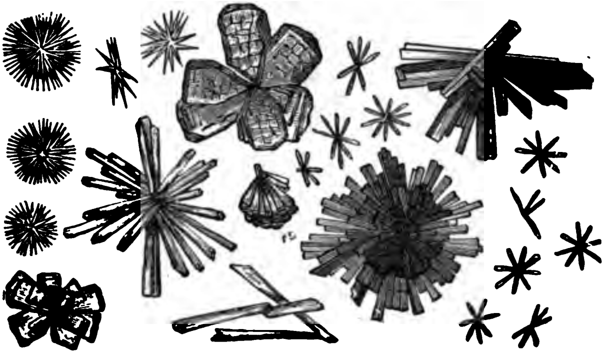


Fig. 97.—Stellar phosphates. (*After Finlayson.*)

(7) **Xanthin** is of extremely rare occurrence; the crystals are said to be similar to "whetstone" crystals of uric acid, but are soluble in ammonia, in warm hydrochloric acid, and in nitric acid.

(8) **Tyrosin** is generally found associated with leucin, but occurs independently also. It forms colourless sheaves of fine needle-like crystals. A similar appearance may be presented by several other *deposits;* therefore, if there be any doubt as to the

nature of the sediment, a chemical analysis may be necessary (Fig. 96, *a*).

(9) **Leucin** occurs in urine as yellow spherical masses without obvious crystalline structure. Leucin and tyrosin occur together in acute yellow atrophy of the liver (Fig. 96, *c*).

In alkaline urine the following occur :—

(1) **Phosphates.**—These may either be salts of phosphoric acid and calcium, or of phosphoric acid with ammonium and magnesium.

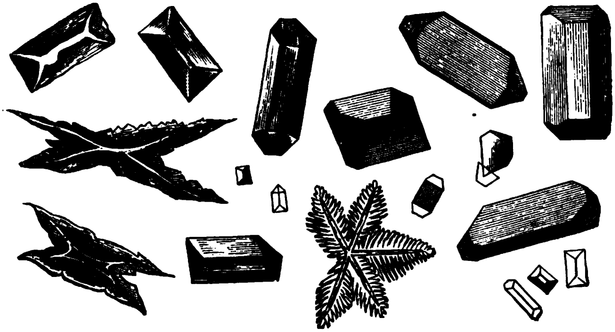


Fig. 98.—Triple phosphates. (*After Finlayson.*)

(*a*) Phosphate of lime is found either in an amorphous or a crystalline form, the latter being also known as **stellar phosphate** (Fig. 97).

Amorphous phosphate of lime occurs in small white granules, as a deposit at the bottom of alkaline urine. To the naked eye the sediment is white and flocculent; unlike urates, it has no affinity for urinary pigment. The deposit is increased on heating.

Stellar phosphates are rather uncommon. They consist of colourless prismatic crystals, which occur either singly or more often in radiating clusters. They are found in very faintly acid as well as in neutral and alkaline urine. Roberts is inclined to regard the presence of this deposit in abundance as an accompaniment of some grave disorder.

(*b*) Ammonium magnesium, or "**triple** " **phosphate**, is deposited in ammoniacal states of the urine. To the naked eye the sediment appears very white, and when the crystals are large they may be visible as bright points. Sometimes the deposit also clings to the sides of the glass and forms a film on the surface of the urine.

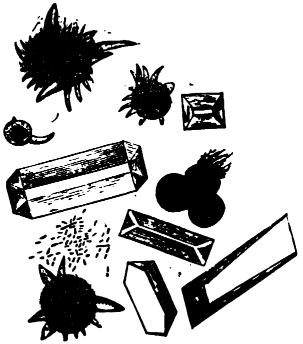


Fig. 99.—Deposit in alkaline fermentation of urine, showing urate of ammonia, triple phosphates, and bacterium ureæ.

The crystals are incomplete, triangular, colourless prisms, which may offer considerable variations in appearance, according to their length and degree of perfection. Often they are described as "knife-rest" or "coffin-lid" crystals. If the ammoniacal change is well marked, and still more if excess of ammonia is added to healthy urine, the deposit takes the form of feathery stars, and is then known as a precipitate of "feathery" phosphates (Fig. 98).

It is difficult to preserve these crystals permanently, but they keep fairly well in a solution of ammonium chloride.

(2) **Urate of ammonia** occurs in alkaline urine, and is very commonly present in cases of cystitis. Microscopically, it occurs in small spherical masses, which are practically indistinguishable in *many* instances from those of urate of soda, except *that they* are generally darker and more opaque, and,

unlike the soda salt, are associated with crystals of triple phosphate. The spheres may have smooth surfaces, or they may be beset with innumerable spiny processes (Fig. 99).

(3) **Carbonates** generally occur in human urine

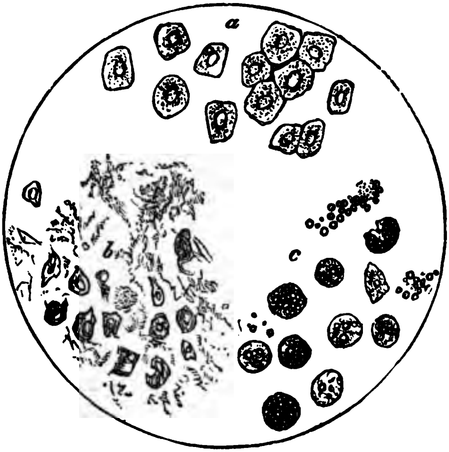


Fig. 100.—Renal epithelium. (*After Roberts.*)
a, normal; *b*, disintegrated; *c*, fatty.

as granular particles, which dissolve in acetic acid with evolution of CO_2. As phosphates give off no gas on solution in acetic acid, it is quite easy to distinguish between them. On rare occasions in human urine, and commonly in horses' urine, carbonate of lime appears in the form of dumb-bells or of spheres with a radiating crystalline structure.

(4) **Cholesterin** has occasionally been found in the urine; it occurs in characteristic thin, rhomboidal, colourless plates, with a notch at one of the corners (*see Fig.* 145, p, 556).

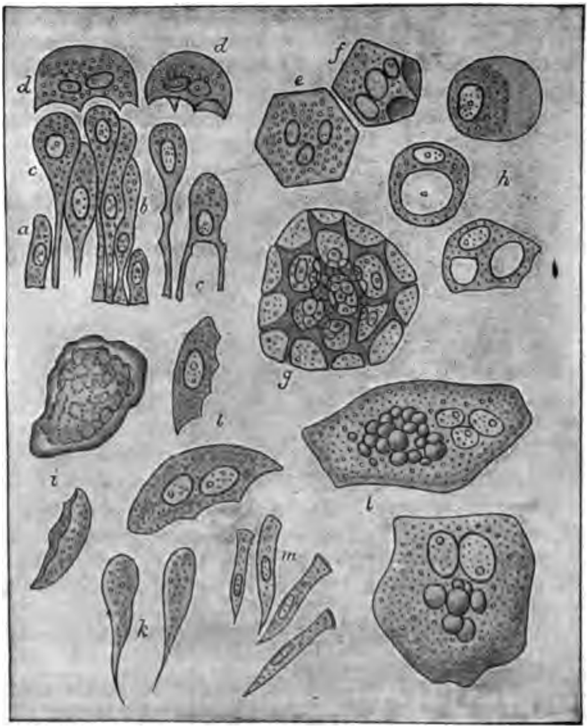


Fig. 101.—Epithelial cells from the urinary passages. (*From Sahli's "Klinische Untersuchungs-Methoden," after Bizzozero.*)

a, cells of the deepest layer; *b*, long cells of the second layer; *c*, "tailed" cells; *d*, flat cells of superficial layer; *e*, *f*, *g*, cells of superficial layer in surface view, with nuclei and indentations; *h*, *i*, *k*, *l*, epithelium from bladder, altered by action of urine; *m*, cells from male urethra.

Other sediments, such as indigo, lime and magnesia soap crystals, and hæmatoidin have been *observed*, but are of little importance.

2.—Organised deposits.

(1) **Red blood corpuscles** are present in cases of hæmaturia, but under certain conditions are rapidly disintegrated, and should therefore be examined for in recently voided urine. According to the density of the urine they appear fairly normal, or swollen, or shrunken and crenated.

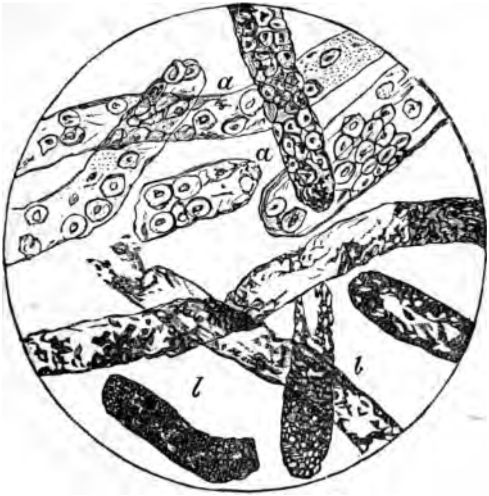


Fig. 102.—Tube casts. (*After Roberts.*)
a, epithelial; *b*, granular.

(2) **Leucocytes and pus corpuscles** occur where there is irritation and suppuration of the urinary tract. According to the length of time which has elapsed, the cells may be indistinguishable from ordinary leucocytes, or they may be very granular and fatty. The addition of acetic acid clears up the cell body and brings two or three nuclei into view. Where pus is present, examine carefully

for pathogenic microbes, especially for gonococci and tubercle bacilli.

(3) In cases of **chyluria** the urine contains nucleated granular corpuscles similar to leucocytes, and very finely divided fatty material, which appears simply granular under the microscope. A few red blood corpuscles are often present. The urine and blood should be carefully examined for the presence of filariæ, particularly if the patient comes from a district where these parasites exist.

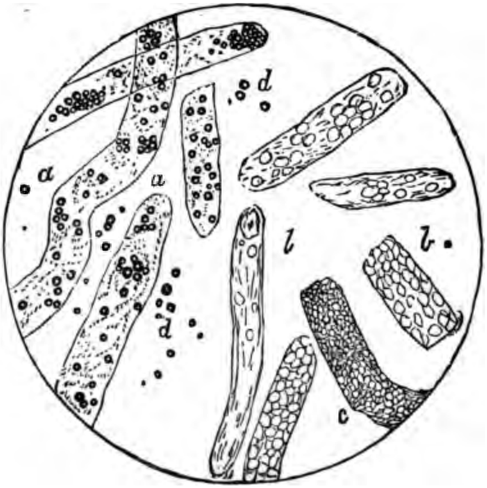


Fig. 103.—Tube casts. (*After Roberts.*)
a, fatty casts; *b*, *c*, blood casts; *d*, free fatty molecules.

In **lipuria**, the fat may occur in larger globules which refract light strongly, and which are sometimes free in the fluid, at other times enclosed in cells or tube casts.

It must not be forgotten that fatty matter may reach the urine unintentionally from an oiled catheter, or may be added purposely in the form of milk by a patient who wishes to deceive the physician.

(4) **Epithelium** from various parts of the urinary tract may be found in the urine. The following varieties are readily recognised :—

(*a*) *Renal epithelium.* This is polygonal, nucleated and rather larger than a leucocyte. It may present fatty degeneration, or be more or less disintegrated (Fig. 100).

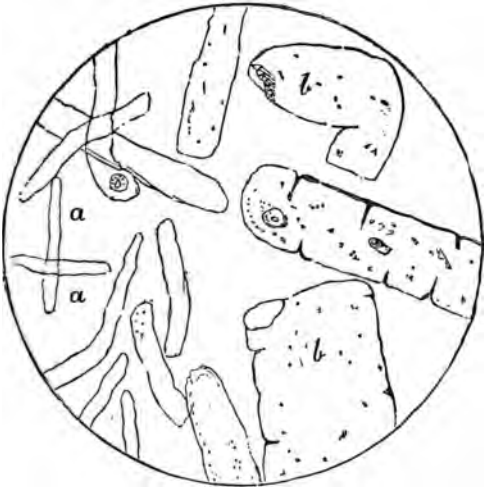


Fig. 104.—Hyaline (*a*) and waxy (*b*) tube casts. (*After Roberts.*)

(*b*) *Epithelium from the bladder and urinary passages* presents various appearances, according to whether it is derived from the more superficial or deeper layers. Formerly, tailed cells were thought to indicate implication of the pelvis of the kidney; this is, however, inaccurate. They may equally well proceed from the deeper layers of the bladder epithelium, as may be seen from the accompanying diagram (Fig. 101).

(*c*) *Vaginal epithelium* is very commonly present in the urine of women. It is squamous, and the large cells appear sometimes singly, at other times in groups.

(5) **Spermatozoa** occur at times in the urine, where their characteristic appearance makes it easy to recognise them.

(6) **Prostatic threads** are found when there is chronic inflammation of the prostate, especially after gonorrhœa. They consist of mucus, and are mostly voided with the first portions of the urine. They are much larger than tube casts, being visible readily enough to the naked eye as they float in the urine or on its surface.

(7) **Tube casts** (Figs. 102, 103, 104).—The following classification, which is more satisfactory than those usually adopted, is given by Prof. Senator, of Berlin.*

There are three main groups of tube casts:—

i. Casts wholly or mostly composed of cellular structures.

ii. Granular casts.

iii. Amorphous casts, having a homogeneous structure, and occasionally striated on the surface.

Group i. **Cellular.**—The cells may be epithelial or composed of red blood corpuscles or leucocytes.

(*a*) Epithelial. The casts may be completely covered with epithelial cells, as though the whole epithelium had scaled off a tubule, or the cells may have been separately detached and subsequently moulded. The cells may or may not show a nucleus, and they may appear fresh, or affected by granular or fatty degeneration.

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* "Die Erkrankungen der Nieren," in Nothnagel's "Specielle *Pathologie und* Therapie," vol. xix.

(*b*) The red blood corpuscle casts exhibit a surface thickly covered with the minute round corpuscles.

(*c*) Leucocytes rarely form casts by themselves, but are pretty often found adhering to the surface of other casts.

Group (ii.) **Granular.**—The granules are sometimes coarse, at other times fine. They are sometimes fatty, at other times they result from granular degeneration of protoplasm. They represent in some instances the relics of broken-down epithelium, in other cases they result from a granular change occurring in old amorphous tube casts.

Group iii. **Amorphous.**—This group contains two varieties the hyaline and the waxy.

(*a*) Hyaline tube casts are pale, transparent, and homogeneous. Occasionally the surface is striated. They may be almost invisible, but are rendered more prominent by the addition of iodine solution. Their origin has been variously accounted for. Senator believes that in the vast majority of cases they are derived from epithelium which has undergone hyaline degeneration, or has yielded a secretion that is coagulable.

(*b*) Waxy casts are broader and more highly refractile than hyaline. Often they are more or less fissured. Possibly they may be formed from other casts which have remained long in the urinary tubules; they are *not* symptomatic of waxy disease of the kidney. Sometimes they give the amyloid reaction with iodine and sulphuric acid, and with methyl violet.

Transition forms between the various groups are not uncommon; often, for instance, a cast is partly epithelial and partly hyaline.

A tube cast frequently picks up adventitious elements from the urine, and thus comes to contain bacteria, or crystals such as oxalate of lime.

In length, tube casts are very variable; occasionally they approach a millimetre in length. One end may be spirally twisted or in rare cases bifurcated. Formerly tube casts were called "fibrin cylinders," but now it is proved that fibrin rarely or never enters into their composition, the only exception being that in the case of red blood corpuscle casts the blood discs may be bound together by a little fibrin.

Structures called **cylindroids** have been described by Thomas and others. They resemble extremely long and narrow tube casts, but are usually considerably flattened. Very varying estimates have been formed of their significance, some observers regarding their presence as quite immaterial, others looking on them with considerable suspicion as being nearly related, both in origin and clinical import, to tube casts. Senator inclines to the latter view.

Not to be confounded either with tube casts or cylindroids are the small strings of mucus which occasionally are present in a urinary sediment. Small clumps of micrococci and short so-called "prostatic threads" are also liable to be misinterpreted by an inexperienced observer. When there is reasonable cause for doubt, the addition of acetic acid or some other reagent will often make the discrimination perfectly simple.

It is frequently difficult **to preserve tube casts** permanently for microscopic examination. The two methods which the writers have found most serviceable are these:—

(*a*) Collect some of the sediment containing the casts, wash rapidly in water, and drop into a conical glass containing picrocarmine stain. When the sediment has collected at the foot of the glass, which will have occurred in twelve hours or so, wash the deposit once more in water, and drop it into a small quantity of Farrant's medium in a conical glass. It will gradually sink in this, and after a day or two samples can be removed from the foot of the fluid, and be permanently mounted in some of the fluid from which they have been withdrawn.

(*b*) Harris proposes the following method: Let urine stand for twelve hours, then remove deposit, and place in a pipette whose upper end is closed by a rubber stopper. The pipette should contain the following preservative fluid:—

Pot. acetate	60 grms.
Chloroform	10 cc.
Distilled water	1,000 cc.

When the sediment has sunk through the fluid it may be transferred to a slide along with a drop of the preservative, and permanently sealed there. Specimens keep fairly well.

The use of a centrifuge is of great value for securing tube casts in perfectly fresh urine, and for washing them rapidly in water prior to preserving them.

(8) Tumours of the bladder, especially when villous, may often be detected by the presence of **fragments** of the growth in the urine. These show a core of connective tissue with its blood-vessels, coated with several layers of nucleated epithelial cells. In cancerous tumours, though their *débris* is commonly enough present, yet nothing at all so characteristic is to be seen as is found in the papillomata.

(9) **Elastic fibres** are often present in cases of ulceration of the bladder. They may be detected either without special treatment, or after the use of caustic soda, as was described in Chapter VI., after a preliminary filtration of the yet acid urine to remove the phosphates, which would otherwise be precipitated copiously when an alkali was added.

The **parasites** that infest the urinary tract are in temperate climates neither numerous nor common. Senator[*] enumerates the following:—Echinococcus, cysticercus cellulosæ, eustrongylus gigas, distoma (or Bilharzia) hæmatobium, filaria sanguinis hominis, nephrophages sanguinarius (a member of the arthopoda related to the acari), rhabditis (sp. ?), and certain psorosperms. Several of these are of such extreme rarity as to be of no practical importance; echinococcus, cysticercus, and filaria having been described elsewhere,* distoma hæmatobium alone needs to be referred to in this chapter.

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[*] *Loc. cit.*, p. 420 *et seq.*

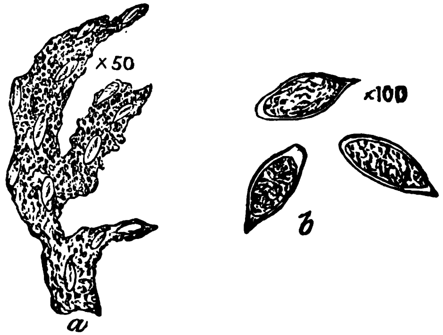

Fig. 105.—Ova of Bilharzia hæmatobium in urine. (*After Roberts.*)
a, × 50 in mucus ; *b*, × 100 in urine freshly voided.

D. hæmatobium. The ova measure 0·12 mm. by 0·04. A spine projects at one pole or at a little distance from it. In urine the spine is usually situated at the pole; the form with a lateral spine predominates in ova obtained from the rectum (Fig. 105).

The adult male Bilharzia is thicker and shorter than the female, and is provided on the ventral surface with a gynæphoric canal. The female is cylindrical and wormlike. The male measures 12 mm., the female about 16 mm., in length. Their habitat is in the blood-vessels of the portal system and in the venous plexuses of the bladder and rectum. The ova escape from the blood-vessels into the tissues of the body. Those which reach the rectum and bladder are discharged, and enable a diagnosis to be made. The parasite is very common in Egypt, where nearly a fourth part of the native population are supposed to suffer from it.

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* Pp. 100 and 218.

After the urine has been voided for some time, it becomes contaminated by numerous non-pathogenic fungi and infusoria; but several pathogenic bacteria occur in the urinary tract, and in cases of doubt should always be sought for. The chief of these are the gonococcus, the tubercle bacillus, which must not be mistaken for the morphologically similar smegma bacillus, and the bacillus coli communis.*

In cases of cystitis a great variety of bacteria may occur. Actinomyces, though rarely present in the kidney, has been found there.

Foreign bodies often occur in urine which has been set aside for examination. Besides hairs, feathers, moth-wing scales, cotton, woollen, and silk fibres, starch grains derived from dusting powders—and readily recognised by their turning blue on the addition of a little dilute tincture of iodine—and, more confusing than any of these, pinewood dust swept from the floor—one occasionally finds fragments of the contents of dermoid tumours or abscesses that have opened into the bladder or ureter. Small shreds of striped muscle may in rare instances be voided with the urine, and are derived, in some cases at least, from a sloughing psoas abscess.

It may happen also that the patient has been sick, and sputum or vomited matter may be more or less abundantly mixed with the urine. If these sources of contamination are forgotten, there is a risk of very erroneous interpretations being given of not a few urinary deposits.

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* *For further information see* Chapter XIV., pp. 575 and 584.

CHAPTER VIII.

THE SKIN.

FOR the examination of the skin and its appendages the patient should be stripped as completely as circumstances permit and placed in a good light.

One should first note the **colour** of the skin as a whole. In anæmia the skin is pale; in chlorosis it has a greenish tint; in pernicious anæmia it is lemon yellow. In order to distinguish the yellowness of pernicious anæmia from jaundice, look at the conjunctiva. The best way to do that is to place one hand on the patient's forehead, ask him to look at the ground, and then raise the upper eyelid with the thumb. In jaundice the conjunctiva is seen to be yellow where it covers the sclerotic; in anæmia it is white. In judging of the degree of anæmia, one should be guided more by the colour of the mucous membranes than by that of the skin itself. The conjunctiva lining the lower eyelid is most usually taken as an index. It is easily seen by getting the patient to look up while one depresses the lower lid with one finger. Instead of being pale, the skin may be abnormally red or *flushed*. The flushing may be general or local. Its exact extent should always be noted, and whether or not it fades on pressure. The best way of telling whether any redness of the skin fades on pressure or not is to place a lens on the skin and press it down. It will then be seen whether or not the skin becomes pale under the lens.

The term **tache cérébrale** is applied to the red flush which appears in some cases of intracranial disease when the skin is stimulated. To elicit its *presence,* draw the finger nail firmly across the patient's forehead. A red line soon develops along the track of the nail, and persists for some time. It is due to a disordered vasomotor supply, but is found in other conditions besides those of cerebral irritation, and is therefore not of much diagnostic value.

Taches bleuâtres are steel-blue spots, which may occur in large numbers, usually on the trunk and thighs. They are deeply placed in the skin, irregular in outline, and without diagnostic significance. They are probably always associated with the presence of pediculi.

Rarer alterations in colour of the skin are those due to the taking of nitrate of silver and those which occur in Addison's disease. The former constitutes what is known as *argyria.* It consists in a leaden grey hue of the whole skin, which is unaffected by pressure. The pigmentation of Addison's disease consists in a *bronzing*, which appears first on parts in contact with the air, and next on those which are exposed to pressure. It is made up of small brownish spots, which fade off at their margins into healthy skin. The lips and buccal mucous membrane should always be examined in cases of supposed bronzing. In Addison's disease they often exhibit marks of pigmentation of a dark bluish-black colour, which have been compared to the stains produced by sucking a pen. Diffuse pigmentation of the skin is also a common occurrence in pregnancy (chloasma), and in many cases of pulmonary tuberculosis, and also after the prolonged administration of arsenic.

Having noted any alteration in the colour of the skin, one should look for the presence of any **eruption.** If any such be observed, the patient should be questioned about it on the lines laid down on p. 10. The exact situation and extent of the eruption should be noted, and whether it is symmetrical or confined *to one side only.* These general facts having been noted, one should pass to a description of the minute characters of the eruption. In order to do this, it must be borne in mind that every cutaneous eruption consists of a "**primary lesion,**" to which secondary lesions may or may not be superadded. The following is a description of the different *primary lesions* which may be met with :—

1. **Macules** or spots.—Any abnormal change in the colour of the skin confined to a limited area. Always note whether or not they fade on pressure. The spots of typhoid fever, for example, fade on pressure, whilst those due to hæmorrhages into the skin, as in the bites of fleas, do not.

2. **Papules.**—Solid projections above the surface, which are not larger than a pea. The term **tubercle** is applied to any solid projection from the skin which is larger than a pea, but not larger than a cherry. Anything larger than that is called a **tumour.** Always note whether the top of a papule is rounded, as in some forms of eczema, pointed as in acne, or flattened as in lichen. As regards the base, observe whether it infiltrates the skin widely or not. The wider the infiltration the more extensive and severe the inflammation.

3. **Vesicles.**—Elevations of the horny layer of the epidermis by transparent or milky fluid which are not larger than a pea. If larger than that they should be described as **bullæ** or **blebs.** Always note whether or not there is an area of redness around the base of a vesicle, for such redness indicates that the vesicle is planted upon an inflamed base, a fact which may be of diagnostic value.

4. **Pustules.**—Small elevations of the skin containing pus. Always observe whether there is much infiltration around them or not.

5. **Wheals.**—Slightly elevated portions of skin, the centre of which is paler than the periphery.

Having stated which of these primary lesions it is that composes the eruption, one should next note whether the lesions are isolated (discrete), or whether they run together. It must also be remembered that an eruption may be made up of more than one kind of primary lesion. Thus papules may be mingled with pustules, or pustules with vesicles, and so on.

Next look for **secondary lesions.** These are either produced mechanically or are the result of changes which take place in the primary lesion in the course of its growth or decline. The commonest secondary lesions of mechanical production are **excoriations** due to scratching and **fissures** (rhagades)—deep cracks going down to or through the corium, and produced by the stretching of the skin after it has become inelastic owing to infiltration. Fissures are often very painful.

The following are the *secondary lesions* produced by changes in those which are primary:—

1. **Desquamation.**—If the primary lesion be a dry one (macules or papules), a mere scaling off of epidermic cells occurs, and the eruption is then said to be "*scaly.*"

In moist lesions (vesicles, pustules, bullæ) the epidermic cells become glued together by the dried fluid, and a *scab* or *crust* forms. The **scab** may be serous, purulent, hæmorrhagic, or sebaceous, according to the nature of the contents of the primary lesion.

2. **Infiltration** may occur around the primary lesions, leading to the production of a leathery feeling in the skin. This is usually the result of prolonged chronic inflammation.

3. **Pigmentation** may occur around the primary lesions. This is also usually due to prolonged inflammation.

4. ***Ulceration.***—Due to breaking down of the

primary lesions and destruction of a part of the true skin.

The points to note in describing an ulcer are (1) the nature of the floor of the ulcer and the granulations covering it; (2) the character of the edge—smooth, raised, undermined, etc.; (3) the discharge—is it serous, purulent, watery, fetid, etc.? (4) the character of the surrounding skin, whether indurated, pigmented, etc.

5. **Scar formation.**—This only occurs where the true skin has been involved, *i.e.* where there has been an ulcer. Describe the scar, noting especially whether it be thin or thick, freely movable or adherent to the deeper tissues, pale or livid, pitted or not, surrounded by a zone of pigmentation or not.

Proceed now to the **palpation** of the skin. Pass the hand gently over it, pinching it up between the forefinger and thumb, and note the following points:—

Is it smooth or rough, thin or thick, dry or moist? If there be any visible sweating, note whether it is general or local; whether it is attended or not by any flushing of the skin; and whether the sweat has any particular odour.

The **elasticity** of the skin should be investigated. If a fold of healthy skin is pinched up it immediately flattens itself out again when released. Sometimes, however, it only does so very slowly, remaining for a considerable time in a creased condition. This indicates a diminished elasticity. It occurs not unfrequently in debilitated and in old persons It may also be often observed in babies exhausted from diarrhœa, and is then believed to indicate interference with the renal functions.

The condition of the **subcutaneous tissue** should also be investigated. It may be infiltrated *with fluid* (œdema), with solid material (as in myxœdema), or with air (subcutaneous or surgical emphysema). The presence of œdema is usually recognised by the fact that if the skin be pressed with the finger, especially over a hard body such as a bone, a pit is left which persists for some little time. It must be borne in mind, however, that this is not an invariable guide. In some cases of œdema no pitting can be produced. This is specially apt to be the case where the œdema is one of very long standing. The best place to look for slight degrees of œdema in cardiac disease is behind the malleoli of the tibia and fibula. In chronic renal disease œdema can often be earliest detected beneath the conjunctiva. This subconjunctival œdema is seen by pushing up the lower lid over the sclerotic. A little drop of fluid resembling a tear is then squeezed up underneath the conjunctiva over the sclerotic.

Subcutaneous emphysema gives rise on palpation to a crackling sensation, which has been compared to that which is experienced in handling a bag of feathers. It starts in, and is usually confined to, the neighbourhood of the air-passages or air-containing organs, and is due to the establishment of an abnormal communication between such a passage or organ and the subcutaneous tissue. In rare cases it may be of bacterial origin.

Microscopic examination of the skin and its appendages is confined to the diagnosis of some parasitic diseases, of which the following are the chief (Fig. 106):—

1. **Scabies or itch.**—This is due to the acarus scabiei. The female acarus is larger than the male, and forms burrows in the skin, in which the eggs are deposited. These burrows should be looked for between the fingers and on the inner aspects of the wrists. They are recognised with the naked eye as little *short dark lines* terminating in a sort of

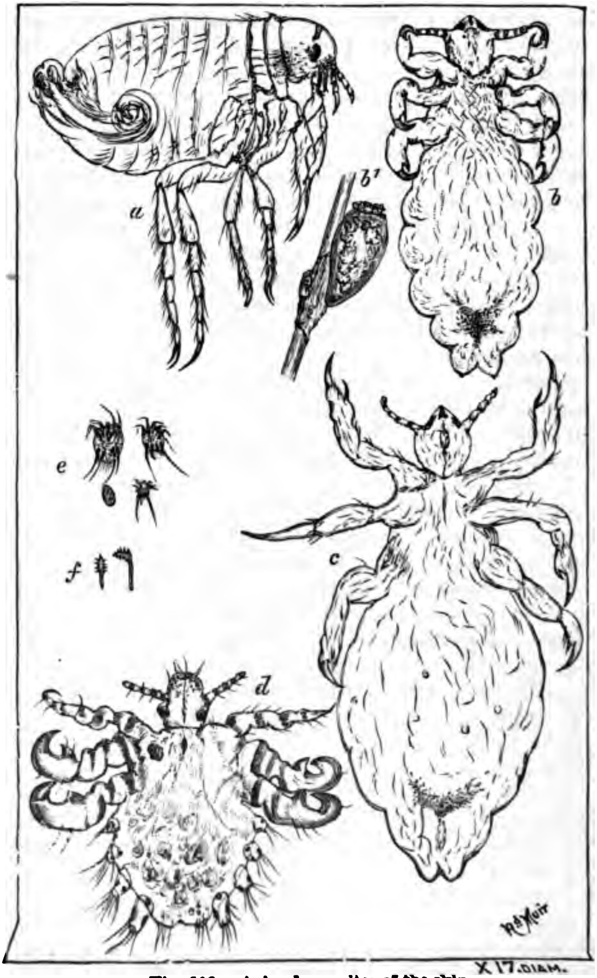


Fig. 106.—Animal parasites of the skin.

a flea; *b*, Pediculus capitis; *b'*, ovum of ditto, with embryo ("nit"); *c*, P. corporis; *d*, P. pubis; *e*, Acarus scabiei (female, male, ovum, larva); *f* Demodex folliculorum.

shining spot of skin. The eggs lie in the dark line; the insect in the shining spot. It may be picked out by means of a flat surgical needle passed along the black line to the clear spot. The use of a lens aids the operation, which is by no means invariably successful, and permits of the recognition of the insect. The latter may be placed on a slide under the microscope for more minute inspection (Fig. 106, *e*).

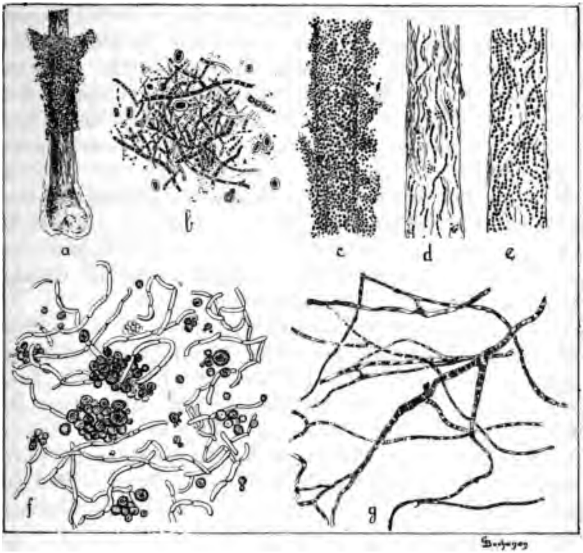
2. **Pediculosis.**—Three varieties of pediculus occur. Pediculus capitis on the head, P. corporis on the trunk, P. pubis on the pubic and axillary hairs. The eggs or "nits" of the P. capitis are stuck on to the hairs (Fig. 106, *b'*). From their position on the hairs one can judge roughly of the duration of the condition, for they are fixed on at first near the root of the hair, and are then carried up with the latter in its growth. The higher up the nits are, therefore, the longer have pediculi been present. The P. corporis should be looked for in the seams of the clothes, especially where the latter come into close contact with the skin—*e.g.* over the shoulders. The bites of the parasite produce hæmorrhagic spots, each with a dark centre and a paler areola. Marks of scratching should always be looked for on parts accessible to the patient's nails.

The microscopic characters of the pediculi are shown in Fig. 106, and require no verbal description. It will be noticed that the P. corporis is the longest and narrowest of the three, the P. pubis is shortest and broadest, and the P. capitis is between the two in size, The P. pubis is also distinguished from the others by being yellowish-brown in colour. P. capitis and P. corporis are both greyish in colour, though the latter varies considerably with the colour of the skin of its host. The shape of the thorax and abdomen forms a distinguishing character between *P. capitis and P.* corporis (Fig. 106, b, c, d).

3. **Ringworm.**—Investigation by Sabouraud and others has shown that two distinct classes of parasites are capable of producing the appearances which are included under the name "ringworm." One of these is not a trichophyton. It goes by the name of *microsporon Audouini*, and is the cause of the commonest and most contagious and intractable form of the disease. Its operations are chiefly confined to the scalp, but 90 per cent. of all cases of ringworm in that situation are due to its presence. The other parasite belongs to the trichophyton family, but it is probable that there are several varieties of it. It is the commonest producer of ringworm of the beard and skin (tinea circinata), but only occurs in about 10 per cent. of the cases of ringworm of the scalp. It succumbs much more readily under treatment than does the microsporon. The trichophyton may be situated mostly outside the hair (*ectothrix* form), or mainly in the hair substance (*endothrix*). Intermediate forms are also described. It is characterised microscopically by having fairly large spores, which are arranged in chains, and the joints of its mycelium are placed at regular intervals. The microsporon has small spores, which are scattered irregularly, and the joints of its mycelium are at unequal intervals (Fig. 107).

A useful method of detecting hairs which are affected by ringworm consists in dabbing over the diseased patch with a piece of wool soaked in chloroform. On evaporation of the latter, the affected hairs are whitened, and look as if covered with hoar-frost. They can thus readily be distinguished from healthy hairs of the same size by the aid of a lens. Hairs affected by favus are not similarly whitened by chloroform.

Microscopic examination.—If one is dealing with a patch of ringworm of the skin, it is sufficient to scrape off some of the scales with a blunt penknife, to place them in a drop of 10 per cent. liquor potassæ, and cover. The mycelium of the fungus will be recognised as branching, refractile threads, amid which the spores are scattered in groups or rows.



ig. 107.—Vegetable parasites of the skin.

a, Favus (Achorion Schönleinii), showing affected hair, × 70; b, Favus (Achorion Schönleinii), showing fungus in a crust, ×290; c, Ringworm (Microsporon Audouini) in hair, surface view, with mosaic of spores, × 200; d, Ringworm (Microsporon Audouini) in hair, optical section, with mycelium, × 200; e, Ringworm (Trichophyton megalosporon) in hair, with spores arranged in chains ("rosary arrangement), × 200; f, Microsporon furfur (from pityriasis versicolor), spores in clusters, × 250; g, Trichophyton megalosporon. Mycelium from a culture, × 250.

If a hair be similarly examined, it will be found to be broken up and full of spores (Fig. 107, *c*). No mycelium can be seen. For diagnostic purposes in simple cases a broken hair should be extracted by broad-pointed forceps, traction being made in the axis of growth so that the bulb may be obtained intact. It should be washed in ether, soaked for a quarter of an hour in liquor potassæ, a drop of glycerine then run under the cover slip, and the edge sealed down with melted paraffin. The spores will be seen in the substance of the hair and in its sheath. Fatty particles are the only thing likely to be mistaken for them. A drop of ether will dissolve fat particles, but leaves the spores unaffected. Liquor potassæ, however, causes the spores to swell. Its use should therefore be avoided if one wishes to distinguish the two varieties of fungus. For that purpose staining is of great help. It should be carried out as follows :—

(1) Soak the hair for some minutes in ozonic ether to bleach it and to remove grease.

(2) Steep for ten to thirty minutes in a mixture of 10 parts of a 5 per cent. alcoholic solution of gentian violet and 30 of aniline water (Appendix, 24).

(3) Dry between blotting paper, and steep in Gram's solution for 2 minutes. This fixes the stain.

(4) Dry again, and soak for 10 minutes in pure aniline to which enough iodine has been added to give it a distinctly brown colour.* This decolorises everything except the fungus.

(5) Wash in pure aniline for a few seconds, then in xylol, and mount in balsam.

Adamson's method of differential staining is briefly as follows :—

Place the hair in a drop of liquor potassæ, and cover. After 20 minutes run in some 15 per cent. alcohol. (This hardens the hair.) Remove the cover glass, wash away any remaining liquor potassæ with more alcohol, mop up the latter, and dry over a flame. Stain in aniline gentian violet for half an hour. Place in Gram for 3 minutes. Decolorise in aniline oil for half an hour. When the fungus is clearly visible, and the hair no longer much stained, remove the oil with blotting paper. Add a drop of concentrated alcoholic eosin, wash off with aniline in a minute, wash off latter with xylol, and mount in xylol balsam.

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* *Nitric acid* may also be used as a decoloriser.

On examination with the high power the two varieties of fungus can be differentiated by the characters already described. It should be noted that the difference in size of the spores of the two varieties is really not very great, after all. The arrangement of the spores and the character of the mycelium are the points to which attention should be directed.

4. **Favus.**—This disease, which is characterised to the naked eye by the production of yellow cup-shaped crusts or *scutula*, is caused by a parasite named, after its discoverer, the Achorion Schönleinii. The hair and accompanying crust may be examined in liquor potassæ, some pressure being exerted on the cover glass in order to flatten out the mass of epidermic scales by which the parasite is apt to be obscured. The hair will be found completely filled with mycelium, and the medullary canal obliterated. The joints of the mycelium are branched, and often present a resemblance in shape to metacarpal and metatarsal bones. The spores are rather large, and arranged in rows or groups (Fig. 107, *a*, *b*).

5. **Tinea versicolor.**—This is produced by the microsporon furfur. A scraping should be examined in caustic potash. The fungus shows a refractile mycelium which interlaces freely, and includes bunches of round spores in its meshes (Fig. 107, *f*). The spores may be stained with saffranin, differentiated by weak acetic acid, and the mycelium counterstained with methylene glue.

6. **Demodex folliculorum** (Fig. 106, f) is a

minute acarus about $\frac{1}{120}$ in. in length, which is sometimes found in the sebaceous contents of comedones. It has a disproportionately large abdomen marked with transverse rings, which give it at first sight the appearance of a minute worm. It possesses a suctorial proboscis and styliform jaws, and from the thoracic portion of the body four pairs of stunted legs project. It is simply a parasite living in sebaceous matter, and is of no pathological importance.

CHAPTER IX.

NERVOUS SYSTEM.

SECTION I.—ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM.

MUCH recent knowledge regarding the structure and mechanism of the central nervous system is still too unsettled to admit of being used at the bedside; but if the student wishes to investigate a case of nervous disease intelligently he must first have a clear grasp of some well-established facts in the anatomy and physiology of the brain and spinal cord. A few paragraphs devoted to these subjects will therefore not be out of place.

1. **Anatomy and physiology of the motor and sensory paths.**—The reader will remember that the motor area of the brain is situated round the fissure of Rolando, the leg centre being highest up, the arm centre next to it, and the centres for the face, lips,

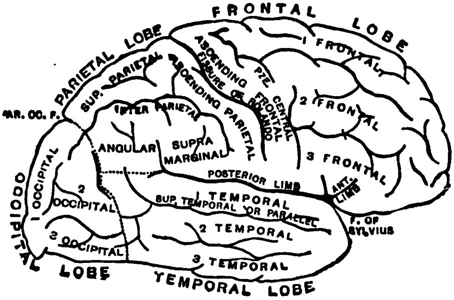


Fig. 108.—*Outer aspect of Right Hemisphere, showing convolutions.*

and tongue being lowest. Stated more exactly, it may be said that the movements of the lower limb are presided over by the upper ends of the ascending frontal and ascending parietal and the posterior end of the marginal convolution; the movements of the upper limb by the middle third of the ascending frontal convolution, the posterior end of the superior frontal, the middle third of the ascending parietal

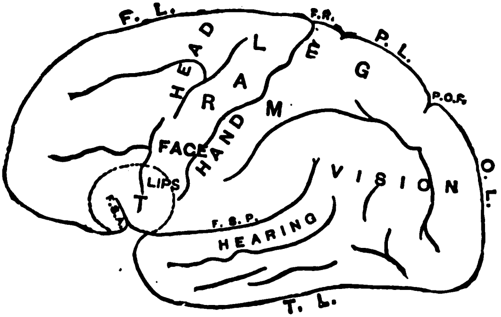


Fig. 109.—Outer aspect of Left Hemisphere, showing functional areas

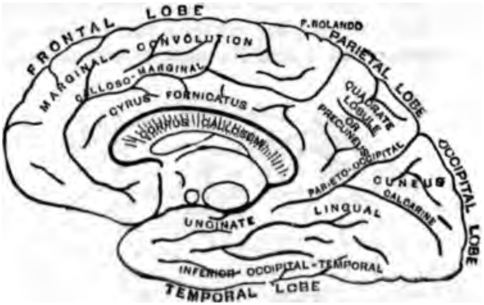


Fig. 110.—Mesial aspect of Right Hemisphere, showing convolutions

(for the hand and wrist), and part of the marginal convolution lying posteriorly to the part which supplies the head. The head, neck, and face are supplied by the lower part of the ascending frontal and by the posterior extremities of the middle and inferior frontal convolutions; also by a small part of the anterior extremity of the marginal convolution. The trunk muscles are supplied by that part of the marginal convolution which lies between the centres of supply for the leg and arm (Figs. 108, 109, 110, 111).

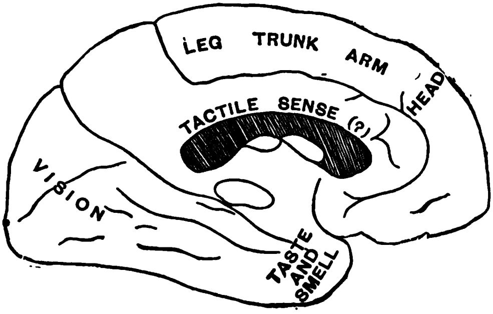


Fig. 111.—Mesial aspect of Left Hemisphere, showing functional areas.

The motor fibres start from the pyramidal cells in the above convolutions and pass in the white matter of the hemispheres to the internal capsule (*i.e.* the knee-shaped band of white matter which is bounded on its outer side by the lenticular nucleus, and on its inner side by the optic thalamus and caudate nucleus). The motor fibres occupy the anterior two-thirds of the posterior limb of the internal capsule, the fibres for the face being farthest forward, those for the leg farthest back, the fibres for the arm being between (Fig. 112). *It is in the internal* capsule that hæmorrhage most frequently occurs, and owing to the close approximation of all the fibres at this point a comparatively small lesion is able to produce a widespread result. From the internal capsule the motor fibres descend to the crus cerebri, occupying the middle third of its anterior aspect. As they descend in the crus the fibres for the leg are to the outer side, the fibres for the face are nearest the middle line, and those for the arm are between the two. Entering the pons, the fibres are no longer quite on the surface, but are covered by a layer of transversely-placed fibres. In the upper part of the medulla they form a well-marked bundle, the anterior pyramid lying quite on the surface. *At the* lower part of the medulla the greater number of the fibres cross to the opposite side, forming what

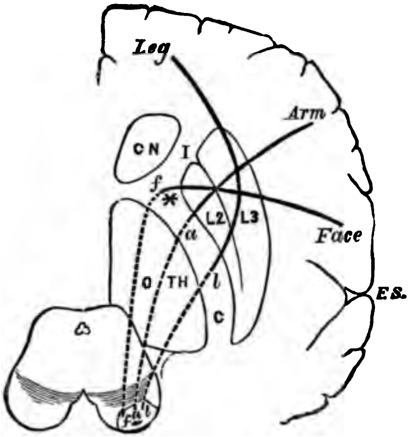


Fig. 112.—Diagram to show relative positions of the face, arm, and leg fibres in their course from cortex to crus. The section through the cortex and crus is vertical; through the internal capsule it is horizontal; * indicates the elbow of the internal capsule.
f, face; *a*, arm; *l*, leg fibres.

is known as the "*decussation of the pyramids*," and run down in the crossed pyramidal tract of the cord to end at different levels in the grey matter of the anterior cornu (Fig. 113). The motor impulses are here transferred to the cells of the anterior cornu and conveyed by their fibres to the anterior roots, and thence to the motor nerves and muscles. The small number of fibres which do not decussate in the medulla are continued down in the anterior or direct pyramidal tract, and end in the anterior cornu of grey matter. Some of them also descend in the crossed pyramidal tract of the same side.

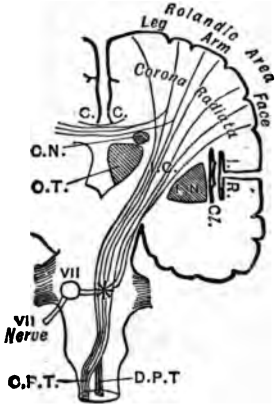


Fig. 113. — Diagram showing the course of the motor fibres from the cortex to the cord.

C C, corpus callosum; C N, caudate nucleus; I R, Island of Reil; C L, Claustrum, * Site of facial decussation; C P T, crossed pyramidal tract. D P T, direct pyramidal tract.

It must also be remembered that a small number of motor fibres do not decussate at all, but end in the anterior cornu of the same side. This, perhaps, explains the fact that after a unilateral central lesion the knee jerk on the same side may be exaggerated as well as that of the opposite leg.

In thus tracing the course of a motor impulse, we have spoken of nerve cells and nerve fibres. It would be better, however, to discard these names in favour of the more recent terminology, which describes a nerve cell, its dendritic processes, and the fibre connected with it (axis cylinder process) as a "**neuron.**" *Thus, anatomically*, the motor impulses are conveyed by means of two neurons. One of these is the pyramidal cell in the cerebral cortex and the motor fibre arising from it (*i.e.* its axis cylinder process) and ending in the anterior cornu. The other is the anterior cornual cell, and the fibre arising from it and ending in the muscle. There is apparently no direct anatomical continuity between these two neurons, but the nerve impulse is able to pass from the one to the other by contact (Fig. 114).

This conception has also the advantage of making clearer some well-known physiological and pathological facts. Thus it can easily be understood that if one part of a neuron be injured the health of the whole neuron suffers. If, for example, what one may call the body of the upper neuron (*i.e.* the cortical cell) be injured, the axis cylinder process of the neuron (*i.e.* the motor fibre) is also affected, and ultimately undergoes the process spoken of as degeneration. Conversely, if a motor

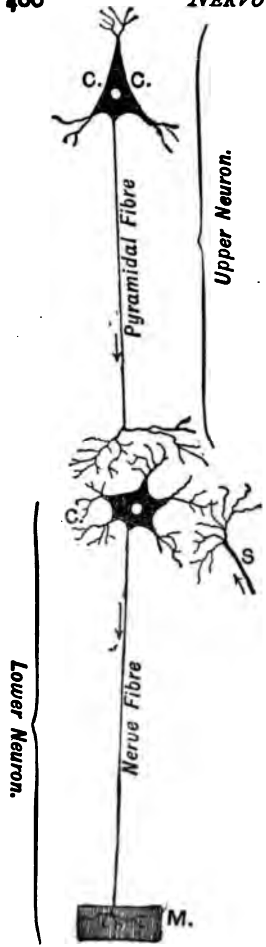


Fig. 114.--Upper and Lower Neurons of Motor path.
C C, cerebral cell; S C, spinal cell; S, sensory fibre; M, muscle.

fibre be cut across—say, in the spinal cord—the health of the cortical nerve cell, of which it is a process, becomes secondarily impaired.

We have spoken of the motor path as consisting of an upper neuron (the cortical cell and its motor fibre) and a lower neuron (the anterior cornual cell and its motor fibre). It must be realised that these are not entirely independent of one another. On the contrary, the upper neuron seems to exercise a sort of restraining influence on the lower, which checks the outflow of nerve energy which is constantly proceeding from the lower neuron to the muscles, and which produce the "tone" of the latter. The result of this is, that if the upper neuron suffers damage in any of its parts, the restraint on the lower neuron is diminished, the tone of the muscles is raised, and, as a consequence, what are known as the "tendon reflexes" of the muscles become exaggerated (*see* p. 478). The influence which the lower neurons exert on muscles in maintaining their tone is sometimes spoken of as a "trophic" influence. Thus, if the lower neuron be injured in any part (*e.g.* if there be inflammation of the grey matter of the anterior cornu, or if a motor nerve be cut across) the corresponding muscle fibres lose their "tone," they become flabby and atrophied, and are then said to be "degenerated."

The course of the **sensory fibres** has not yet been definitely made out. The fibres for pain and heat sensations seem to run together and to enter the grey matter shortly after reaching the cord. They then cross to a large extent to the opposite lateral column. The fibres for touch and cold do not appear to decussate so soon. They may perhaps run in the artero-lateral tract. The fibres of the posterior columns end in the nucleus gracilis and nucleus *cuneatus of the* medulla. Up to this point the neurons concerned have their cell elements in the posterior root ganglia, which are therefore said to be "trophic" for the sensory fibres from the periphery up to the medulla.

There a new set of neurons begins with the cells of the nucleus gracilis and nucleus cuneatus. The fibres arising from these cross to the opposite side in the lower part of the medulla, forming the decussation of the fillet, that being apparently the main sensory crossing. They then pass up to the tegmentum, and the majority end in the thalamus. The remainder, however, appear to pass up through the posterior part of the posterior limb of the internal capsule to find their termination in the cerebral cortex. The central convolutions, and especially the gyrus fornicatus, seem to be the parts of the cortex chiefly concerned in the appreciation of sensory impressions, but it is probable that the sense of touch is located in the Rolandic area as well. The muscular sense seems to be located in the deeper layers of the motor cortex. The course of the fibres and the position of the centres for the special senses are described under the section dealing with the cranial nerves. The speech centres and their connections are described at p. 418.

2. The spinal cord.

The cord extends as far down as the interspace between the first and second lumbar spines; the membranes are continued down as far as the body of the second·sacral vertebra.

The *cervical enlargement* reaches to the seventh cervical spine. Its largest part is opposite the disc between the fifth and sixth cervical vertebræ.

The *lumbar enlargement* lies opposite the three lowest dorsal spines, its widest part corresponding to *the body* of the twelfth dorsal vertebra.

Physiologically the cord is to be regarded as made

ROOTS.	LEVEL OF SURFACE ORIGIN.	POINT OF EXIT.
C. I.	Just above arch of atlas.	Between atlas and occiput.
II.	Ranges from just above to just below spine of atlas.	Above axis.
III.	At or just above spine of axis.	Above 3rd cerv.
IV.	From spine of axis to spine of 3rd cervical.	Above 4th cerv.
V.	Ranges from lower edge of spine of axis to that of 4th cervical.	Above 5th cerv.
VI.	Ranges from lower edge of spine of 3rd to that of 5th cervical.	Above 6th cerv.
VII.	Ranges between top of 4th to bottom of 6th spines.	Above 7th cerv.
VIII.	Ranges between top of 5th to top of 7th spines.	Above 1st dorsal D.
D. I.	Ranges between above 6th and below 7th spines.	Between 1st and 2nd D.
II.	Ranges from lower edge of 6th cervical spine to that of 1st dorsal	Between 2nd and 3rd D.
III.	Ranges from upper edge of 7th cervical spine to lower of 2nd dorsal.	Between 3rd and 4th D.
IV.	Ranges from top of 1st dorsal spine to that of 3rd dorsal.	Between 4th and 5th D.
V.	Ranges from top of 2nd to top of 4th dorsal spine.	Between 5th and 6th D.
VI.	Ranges from lower edge of 2nd dorsal spine to upper of 5th.	Between 6th and 7th D.
VII.	Ranges from top of 4th to bottom of 5th dorsal spine.	Between 7th and 8th D.
VIII.	Ranges from top of 5th to top of 6th spine.	Between 8th and 9th D.
IX.	Ranges from between 5th and 6th spines to top of 7th.	Between 9th and 10th D.
X.	Ranges from lower edge of 6th to upper of 8th.	Between 10th and 11th D.
XI.	Ranges from top of 7th to top of 8th spine.	Between 11th and 12th D.
XII.	Ranges between top of 8th and bottom of 9th spine.	Between 12th D. and 1st L.
L. I.	Ranges between top of 9th spine and bottom of 10th.	Between 1st and 2nd L.
II.	Ranges between 9th and 11th dorsal spines.	Between 2nd and 3rd L.
III.	Ranges between top of 10th and bottom of 11th spine.	Between 3rd and 4th L.
IV.	Ranges between bottom of 10th and top of 12th spine.	Between 4th and 5th L.
V.	Ranges between top of 11th and top of 12th spine.	Between 5th L. and 1st S.
S. I.	Ranges between lower border of 11th dorsal spine and top of 1st lumbar.	Between 1st and 2nd S.
II.	Usually between 12th dorsal and 1st lumbar spines.	Between 2nd and 3rd S.
III.	Usually between 12th dorsal and 1st lumbar spines.	Between 3rd and 4th S.
IV.	Usually between 12th dorsal and 1st lumbar spines.	Between 4th and 5th S.

up of a series of superimposed segments, from each of which a pair of nerve roots arises. To enable us to localise focal lesions of the cord it is necessary to be acquainted with the functions of each segment, and therefore with the area of supply of the pair of nerve roots arising from it.

The table on p. 403 shows the points of origin and emergence of the nerve roots from the cord and spinal canal. Plates VI. and VII. exhibit in a diagrammatic way the motor functions of the cervical and lumbar segments and the reflexes over which each segment presides. The sensory functions are exhibited in Figs. 115, 116, and 117.

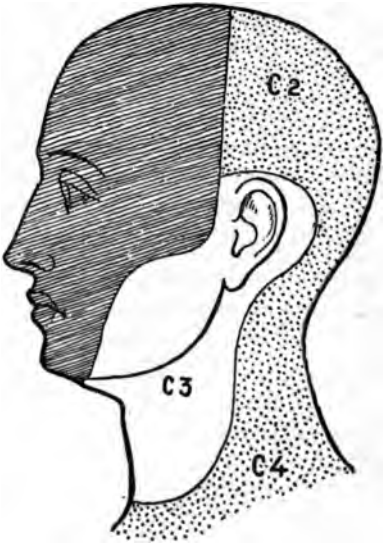


Fig. 115.—Lateral view of the skin areas supplied by the second, third, and fourth cervical segments.

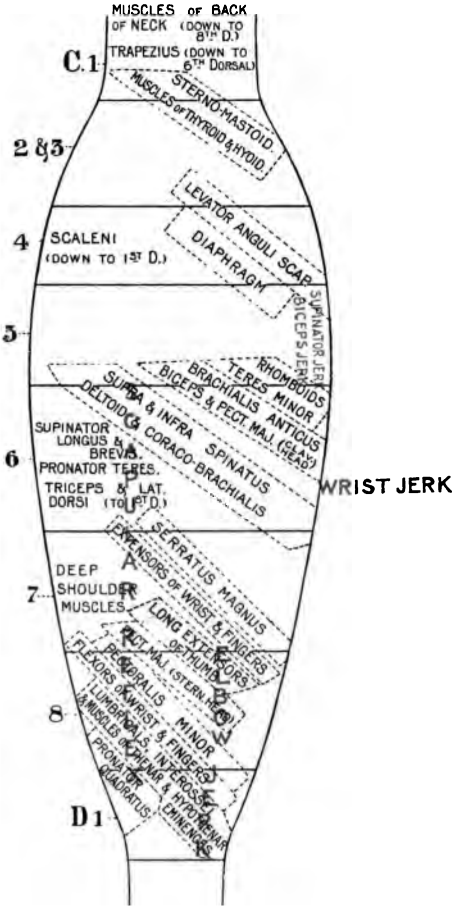


PLATE VI.—MOTOR SEGMENTAL FUNCTIONS OF THE CERVICAL ENLARGEMENT.

[To face p. 45

Figs. 116 and 117 have been constructed by Head mainly from the study of cases of Herpes Zoster. He believes that the areas here shown represent the extent to which one set of fibres of the posterior root ganglion innervate the skin of the body. The supposed relation between any one of these skin areas and a single posterior root ganglion has been confirmed in several cases by post-mortem examination. [Head and Campbell.]

Their importance lies in Head's hypothesis that it is these areas, rather than the full extent of skin supplied by the fibres of a posterior root, that become tender in consequence of disease of an internal organ innervated from the same segment of the nervous system.

It must not be supposed, however, that single areas appear commonly in consequence of visceral disease. The wide extent of skin found to be tender can be analysed by means of these figures into its component elements. Single areas with well-marked limits occur with extreme rarity, and are of scientific rather than of practical interest.

Fig. 118 shows the position of the different **tracts of the cord** on transverse section.

The following list (p. 408) shows the **nerve supply of the muscles** of the trunk and limbs. It may be found convenient for reference in the study of cases of peripheral paralysis. The supply of the head is considered along with the cranial nerves.

The peripheral distribution of the chief **sensory nerves** is sufficiently indicated in Figs. 119, 120, and 121.

Vascular supply of the brain and spinal cord.—The *brain* is supplied by the internal carotid and vertebral arteries. Owing to the position of origin of the left common carotid, an embolus can enter it more easily than it can the artery of the opposite side. Embolic lesions are therefore more frequent in *the left than in* the right cerebral hemisphere.

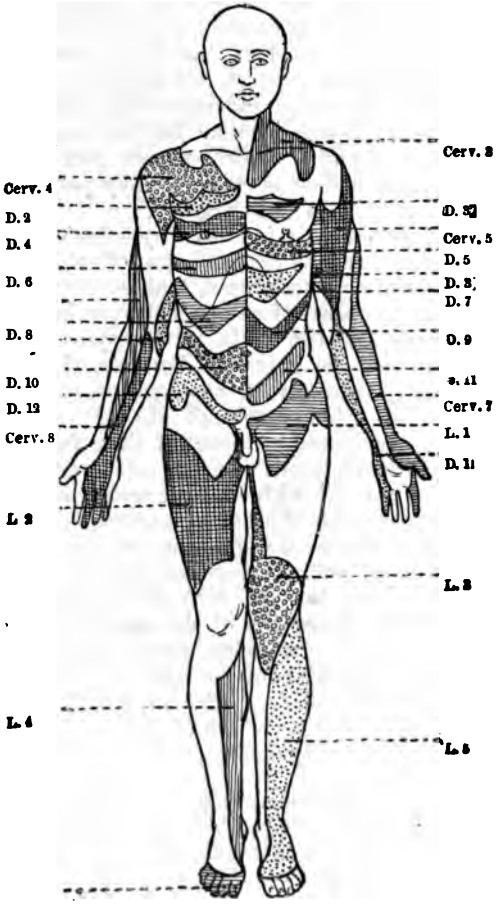


Fig. 116.—Sensory segmental functions of the spinal cord. (*Head.*)
(*See* p. 405.)

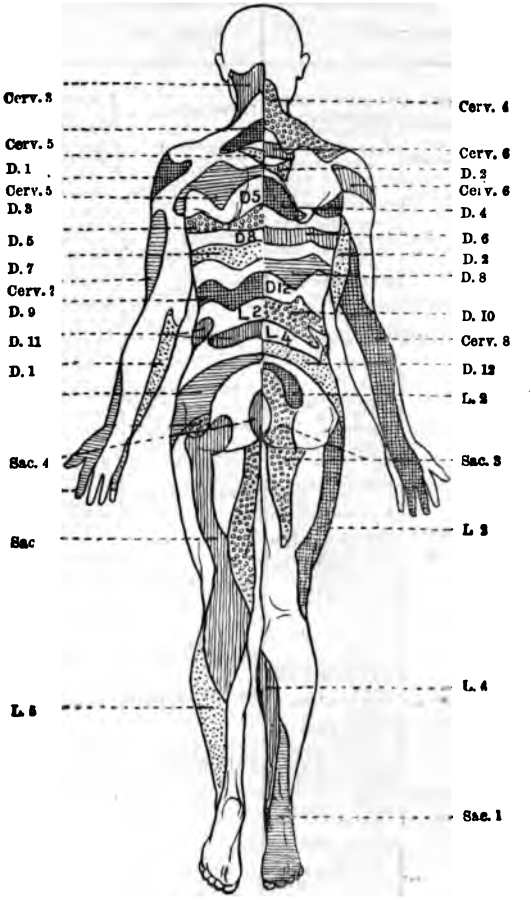
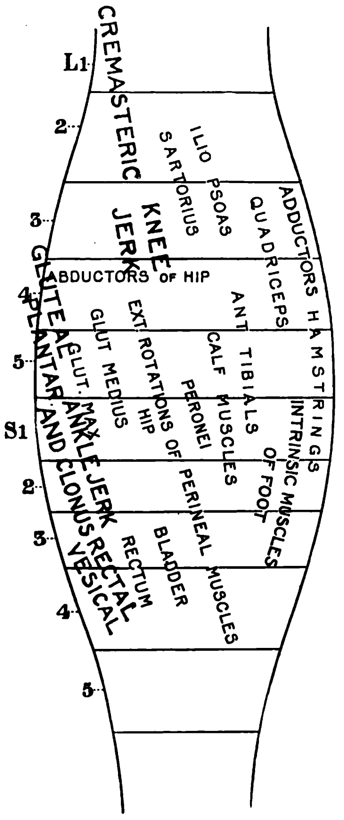


Fig. 117 - Sensory segmental functions of the spinal cord. (Head.)
(See p. 405.)

UPPER LIMB.	
POST. THORACIC ..	Serratus magnus.
SUPRASCAPULARS	Supra-spinatus. Infra-spinatus.
EX. ANT. THORACIC	Pectoralis major (upper part).
INT. ANT. THORACIC	Pectoralis major (lower part). Pectoralis minor.
MUSCULO-CUTANEOUS	Coraco-brachialis. Biceps. Brachialis anticus.
SUBSCAPULAR ..	Subscapularis. Teres major. Latiss. dorsi.
CIRCUMFLEX ..	Deltoid. Teres minor.
MUSCULO SPIRAL	Triceps. Ext. carp. rad. long. Supinator long. B. anticus. Anconeus.
POST-INTEROSSEUS	Supinator brevis. Ext. carp. rad. brev. Ext. carp. uln. Ext. comm. digit. Ext. ossis metac. poll. Ext. primi. intern. poll. Ext. secund. intern. poll. Ext. indicis. Ext. minimi digiti.
MEDIAN	Pronator radii teres. Palmaris longus. Flexor carpi radialis. Flexor sublim. digit. Flexor longus pollicis. Opponens pollicis Abductor pollici
MEDIAN & ULNAR (jointly).	Flex. profund. dig. Flexor brevis pollicis.
ULNAR..	Flexor carpi ulnaris. dductor pollicis - Muscles of little finger. Interossei.

TRUNK AND LOWER LIMB.	
INTERCOSTALS ..	Intercostals. Rectus abdominis. External oblique.
BRANCHES OF LUMBAR NERVES	Erector spinæ. Quadratus lumborum.
GENITO-CRURAL..	Cremaster.
ANTERIOR CRURAL	Sartorius. Pectineus. Rectus femoris. Vastus externus. Vastus internus. Crureus.
OBTURATOR ..	Gracilis. Obturator externus Adductor longus. Adductor brevis. Adductor magnus (with sciatic).
INFER-GLUTEAL	Gluteus maximus.
SUP-GLUTEAL ..	Gluteus medius and minimus. Tens. vag. femoris.
GREAT SCIATIC	Biceps femoris. Semitendinosus. Semimembranosus. Adductor magnus (with obturator).
INT. POPLITEAL	Gastrocnemius. Soleus. Tibialis posticus. Flex. comm. digit. Flex. long. hallucis.
INT. PLANTAR ..	Flex. brev. hallucis. Flex. brev. digit. Abductor hallucis.
EXT. PLANTAR ..	Adductor hallucis. Interossei.
EXT. POPLITEAL	Tibialis anticus. Ext. prop. hallucis. Ext. digit. longus. Peroneus longus. Peroneus brevis. Extens. brev. digit



I.—MOTOR SEGMENTAL FUNCTIONS OF THE LUMBAR ENLARGEMENT.

[*To face p.* 409

The two **vertebral arteries** unite at the lower border of the pons to form the **basilar**, which runs up the middle of the anterior surface of the pons, and ends by dividing into the two posterior cerebrals. It gives off lateral branches which run out transversely over the pons, and vertical branches which pass into its substance. The latter not unfrequently become *thrombosed.*

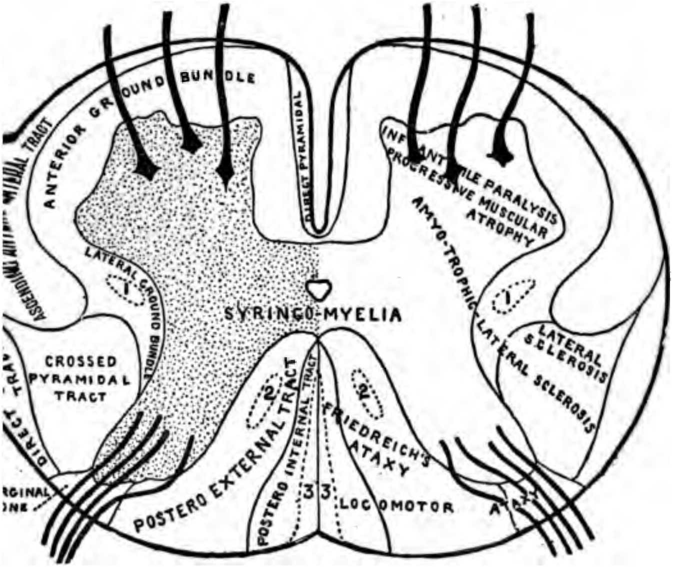


Fig. 118.—Scheme of a transverse section of the spinal cord, showing on the left side the positions of the various tracts, and on the right side the names of the diseases affecting each part.

Descending fibres of fillet in lateral ground bundle; 2, Schultze's descending tract in postero-external tract; 3, Septomarginal tract of Bruce and Muir in postero-internal tract. Besides the names given on the diagram, the following synonyms should be noted:—Postero-internal tract = Fasciculus gracilus = Column of Goll: Postero-external tract = Fasciculus cuneatus = Column of Burdach; Marginal zone = Lissauer's tract; ascending Antero-lateral tract = tract of Gowers; Schultze's tract = "Comma" tract.

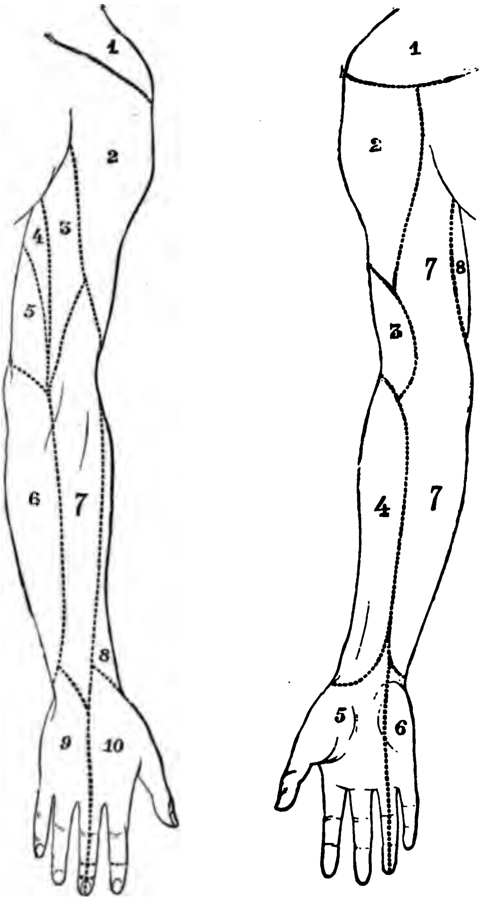


Fig. 119.—Cutaneous nerve supply of upper limb.

Anterior aspect: 1, cervical plexus; 2, circumflex; 3, ext. cut. of musc. spiral; 4, ext. cutaneous; 5, median; 6, ulnar; 7, int. cutaneous; 8, nerve of Wrisberg. *Posterior aspect:* 1, cervical plexus; 2, circumflex; 3, int. cut. of musc. spiral; 4, intercosto-humeral; 5, nerve of Wrisberg; 6, int. cutaneous; 7 ext. cut. of musc. spiral; 8, ext. cutaneous; 9, ulnar; 10, radial.

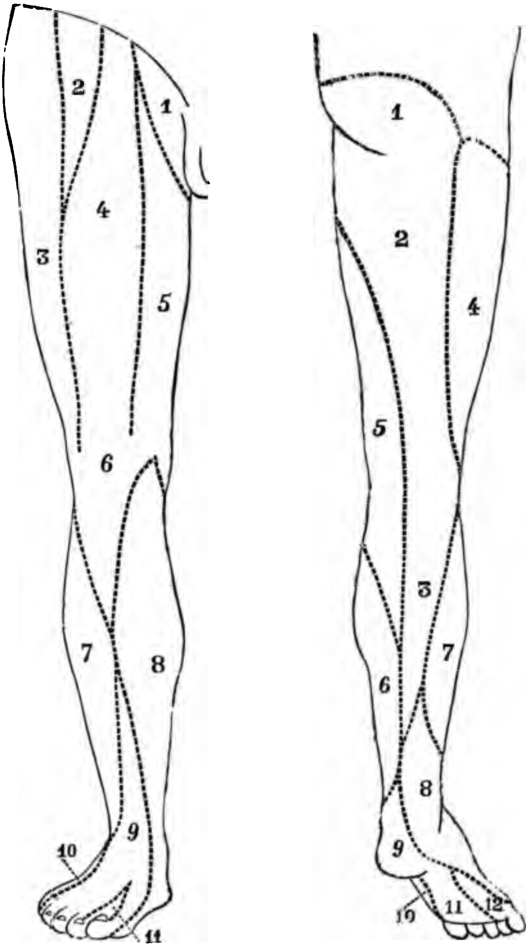


Fig. 120.—Cutaneous nerve supply of lower limb.

Anterior aspect: 1, ilio-inguinal; 2, genito-crural; 3, ext. cutaneous; 4, middle cutaneous; 5, internal cutaneous; 6, patellar plexus; 7, branches of external popliteal; 8, internal saphenous; 9 musculo-cutaneous; 10, external saphenous; 11, anterior tibial. *Posterior aspect:* 1, 2, 3, small sciatic; 4 external cutaneous; 5, internal cutaneous; 6 internal saphenous; 7, branches of external popliteal; 8, short saphenous; 9, posterior tibial; 10, internal saphenous; 11, internal plantar; 12, external plantar.

The **posterior cerebrals** supply the occipital lobes, the lower part of the temporosphenoidal lobes, with the uncinate gyrus, the inner part of the crus and the corpora quadrigemina, and the posterior part of the posterior limb of the internal capsule. Blocking of one of these arteries will therefore involve the visual centre and the sensory fibres.

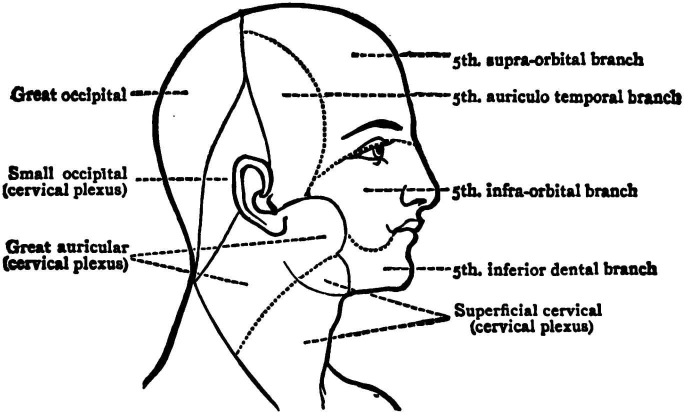

5th. supra-orbital branch
Great occipital
5th. auriculo temporal branch
Small occipital (cervical plexus)
5th. infra-orbital branch
Great auricular (cervical plexus)
5th. inferior dental branch
Superficial cervical (cervical plexus)

Fig 121.—Distribution of the sensory nerves of the head. Compare with it the segmental distribution as shown in Fig. 115.

The basilar artery supplies the upper surface of the cerebellum; the vertebrals supply its lower surface.

The **internal carotid** gives off the **anterior cerebral** artery, which curves round the anterior end of the corpus callosum, and is chiefly distributed to the inner surface of the cerebral hemisphere as far back as the parieto-occipital fissure. It also supplies the superior frontal convolution.

The internal carotid is practically continued on to the brain as the **middle cerebral**, which lies in the

Sylvian fissure. An embolus which has found its way into the internal carotid, therefore, usually ends in the middle cerebral or one of its branches. The middle cerebral gives off *cortical branches*, which supply the motor area and the upper part of the temporosphenoidal lobe. These branches anastomose freely with those of adjoining arteries, hence blocking of one of them may be largely compensated for by the establishment of a collateral circulation. It also gives off *central branches*, which run more or less vertically upwards, penetrating into the brain substance and supplying the basal ganglia. There are two chief groups of these two central arteries—an anterior group called the *lenticulo-striate*, and a posterior group, the *lenticulo-optic*. These arteries are very commonly the seat of miliary aneurysms, and as the lenticulo-striate are more directly exposed to the force of the wave of arterial blood, they are more frequently ruptured than are the lenticulo-optic. These central arteries do not anastomose with one another. They are, therefore, to be regarded as end-arteries. Hence it is that a lesion of one of them is much less likely to be compensated than is a lesion of a cortical branch.

The venous blood from the brain is poured into the **venous sinuses.** Owing to the slow current in these, thrombosis readily occurs in them. The blood from the interior of the lateral ventricles is chiefly returned by the veins of Galen, which end in the straight sinus. Owing to their long course, these veins are frequently exposed to pressure by tumours, etc. This is apt to lead to increased exudation of fluid into the lateral ventricles.

The **arteries which supply the spinal cord** have a long and tortuous course. This renders them liable to thrombosis, but makes embolism of them *almost* impossible. The lower end of the cord is far removed from the point of origin of the vessels which supply it. Hence it is, perhaps, that this part is more liable to suffer damage from nutritional changes than are the higher regions.

The student may now pass to the method of examining a patient with nervous disease, as described in the subsequent sections. We would recommend him to begin by ascertaining the state of the intellectual faculties of the patient, including speech (Section II.). He should then rapidly test the condition of the cranial nerves in their order. How this is to be done is described in Section III. (p. 422). By proceeding thus, valuable information is gained at the outset, which may guide one in his subsequent investigations. The motor, sensory, reflex, and trophic functions should then be examined in order, following the methods described in Sections IV. (p. 457), V. (p. 468), VI. (p. 472), VII. (p. 484). Lastly, the electrical reactions of the muscles and nerves should be tested in those cases in which it may seem necessary (Section VIII, p. 486).

SECTION II.—INTELLECTUAL FUNCTIONS.

It is important to arrive at some idea of the patient's intellectual state early in the taking of a nervous case, as it affords indications that are of help in the subsequent investigation of his symptoms. For example, if one finds that his memory is deficient, one attaches only a limited value to the account that he gives of the onset of his illness or the state of his previous health. Or if one discovers that he is comatose, or unable to understand speech, it is evident that one cannot expect to make much of any attempt to investigate the state of his sensory functions. *This section* will, therefore, be devoted to methods of *investigating* a patient's mental condition, including the functions concerned in producing and interpreting speech.

The first thing to be determined is whether we are dealing with a **right-handed or a left-handed patient.** The importance of this depends upon the fact that right-handed people are left-brained, and *vice versâ*. Ask the patient, if a male, which hand he uses to throw a stone or to pull a cork : if a female, which hand is employed in combing the hair. It is of comparatively little use to ask which hand he writes with, as all children are taught to write with the right hand.

The state of the **memory** next calls for investigation. It may be tested by asking the patient what day of the week it is, what he ate at breakfast, and so on.* Inquire as to his **sleep,** and whether or not he is troubled with dreams.

Note whether or not he is more **emotional** than is normal. An abnormal emotional state is evidenced by the patient's bursting into laughter or into tears on very slight provocation, or by his giving way easily to fits of anger.

In the course of taking his case, one will already have arrived at a general notion of the degree of the patient's intelligence. Sometimes it is necessary to ascertain whether he is the subject of **hallucinations** or **delusions.** An hallucination consists essentially in an imaginary sense impression. A delusion is purely intellectual. It is an erroneous idea which would be incredible to the patient's equals, and which is unshaken by facts. If the patient says he hears voices when no one is present, or if he sees a tree and believes it to be a man, he is the subject of an hallucination—in the former case auditory, in the latter visual. If he declares that he is the Emperor of Russia, he is the victim of a delusion. The existence of hallucinations and delusions is often difficult to ascertain. Sometimes they are discovered by chance; in other cases they can be elicited by skilful questioning; often they are reported by the friends.

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* It is important to distinguish between (*a*) the memory of recent events and (*b*) the memory of older occurrences. Both *should be tested* in every case.

Delirium or **coma** may be present; in such a case the investigation of the intellectual faculties already described is futile.

One should next proceed to the investigation of the **speech functions.***

Supposing that the patient is able to speak, one should note whether there is any peculiarity in his articulation. The following are the chief abnormalities which may be present:—

1. **Stammering.**—This requires no special description.

2. **Lalling,** or baby speech. Ask the patient to read something aloud. If he lalls, one will recognise that all the difficult consonants are dropped; he speaks like a baby, and, if a child, may perhaps make use of words of his own invention. P, B, and M, T, D, and N, are the easiest consonants; K, G, S, Sh, and Ch,. are more difficult; C and L are the most difficult of all. Thus, such a patient has no difficulty in saying "papa," "mamma"; but if asked to say "British constitution," he will probably pronounce it "Bitte tontitu.†"

3. **Scanning or staccato speech.**—The patient speaks slowly and deliberately, syllable by syllable, as if scanning a line of poetry. Ask him to say "artillery." He will pronounce it "ar-til-ler-y."

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* In our description of the methods of clinically investigating the speech functions we have followed very closely the teaching of Prof. Wyllie (*vide* his valuable work on the "Disorders of Speech").

† An aggravated form of lalling in which the patient seems to speak a language of his own is described as idio-glossia.

This is the kind of speech found in cases of multiple cerebro-spinal sclerosis.

4. **Slurring speech.**—The syllables are slurred together as in a state of intoxication. Thus, "British constitution" becomes "Brizh conshishushon." This kind of speech is met with very typically in general paralysis of the insane.

5. **Syllable-stumbling.**—In this condition the patient misplaces some of the letters in a word, and reiterates some particular syllables. Thus, "West Register Street" becomes "West Regigis̄trerer Street."

If the patient's defect consists not in any perversion of articulation, but in an inability to produce speech at all, or to understand it when spoken or when written, then his condition is described as one of **aphasia.**

In order to understand the method of investigating a case of aphasia, it must be remembered that for purposes of speech we have (1) a producing mechanism. This consists of two parts—one concerned in the production of spoken speech, the other in the production of written speech. (2) A receiving mechanism. This also consists of two parts—one for the reception of spoken speech, the other for the reception of written speech.

We may thus classify cases of aphasia as follows:—

1. Lesions of productive mechanism (motor aphasia).	Aphemia (loss of power of talking). Agraphia (loss of power of writing).
2. Lesions of receptive mechanism (sensory aphasia).	Auditory (word deafness). Visual (word blindness)

It must be borne in mind, however, that it is the exception to meet with a case of aphasia of a pure type. Thus, a patient may have both aphemia and also word-deafness. He may be unable to read as well as unable to write, and so on.

The *cortical centres* for the production and reception of speech are situated in the left cerebral hemisphere in right-handed persons, in the right hemisphere in the case of those who are left-handed. Hence the importance of ascertaining early in the investigation of a nervous case whether the patient is right- or left-handed.

The centre for spoken speech occupies the posterior extremity of the third frontal convolution (Broca's convolution), and the lower end of the ascending frontal and probably also of the ascending parietal convolution.

The centre for the production of written speech is believed to be in the posterior end of the second frontal convolution.

The centre for the reception of spoken speech is in the posterior half of the superior temporo-sphenoidal convolution, and that for the reception of written speech (visual speech centre) in the angular gyrus.

The visual speech centre is connected by special fibres with the primary visual centre in each occipital lobe. Hence, a lesion in the left occipital lobe does not produce word-blindness unless it be so situated as to cut off also the fibres which connect the visual centre in the right occipital lobe with the left angular gyrus (Fig. 122, p. 419).

For practical purposes it is best to proceed with

REFERENCES TO FIG. 122.

a, cuneus; c c, posterior extremity of corpus callosum; F 3, Broca's convolution (speech centre); T 1, superior temporo-sphenoidal, or Wernicke's convolution (auditory word centre); 1 and 1′, left and right optic radiations; 2, fibres connecting left angular gyrus with left cuneus and through the corpus callosum (2′ and 2″) with the right cuneus; a lesion at x cuts these fibres as well as the optic radiation, and therefore causes right lateral homonymous hemianopsia—word blindness—but no agraphia; 3, fibres connecting angular gyrus with Wernicke's convolution. The straight black line (4) represents the connection between Broca's and Wernicke's convolutions; the black line bifurcating in front (5) represents the connections of the angular gyrus with the motor region of the left (5′) and right (5″) *hemispheres.*

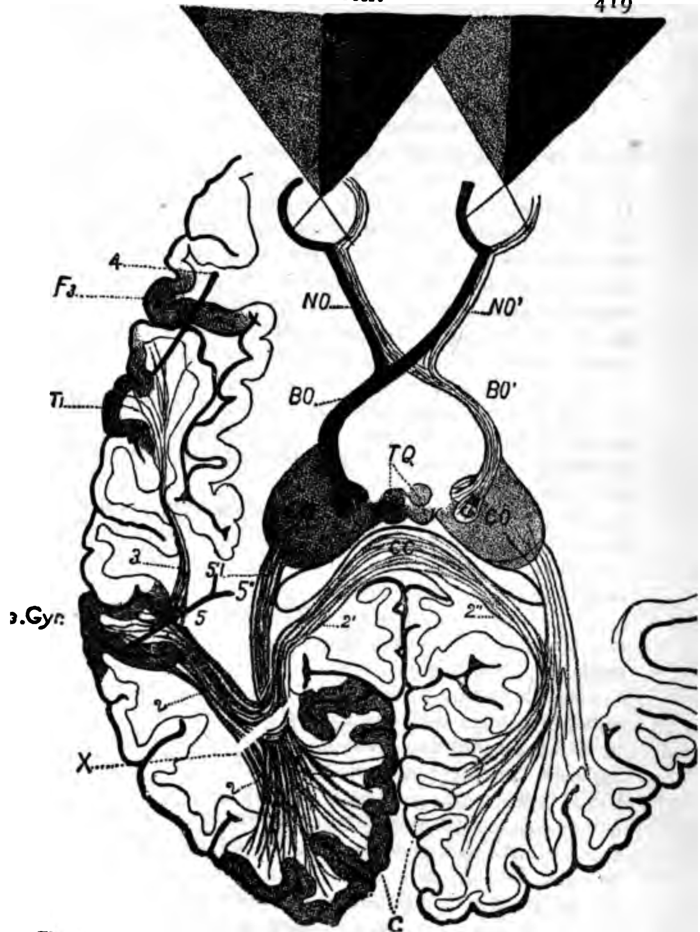

Fig. 122.—Schematic figure, showing the course of the optic fibres. (*From Wyllie, after Déjérine.*)

[For references, see foot of p. 418.]

the investigation of any case of aphasia in the following order:—

I. **Spoken speech.**

1. *How is it received and interpreted?*—Find out, firstly, whether the patient's hearing is good. If so, ask him to put out his tongue, shut his eyes, etc. If he does so, test him as to his understanding of nouns by asking him to touch his nose, ear, chin, forehead, etc., in turn. Then test his verbs by asking him to smile, to whistle, etc. If the patient responds satisfactorily to these questions, he has evidently no difficulty in interpreting the meaning of words heard—*i.e.* there is no *word deafness.*

2. *How is it produced?*

(1) If the patient can only use a few words, make a note of what these are. If he repeats any word or phrase again and again (**"recurring utterance"**), note what it is.

(2) If he has a considerable vocabulary, (*a*) make a note of any examples of lalling, slurring, etc., as described on pp. 416 and 417. This affords an indication of his *power of articulation.*

Test him with such words and phrases as "British constitution," "West Register Street," "Biblical criticism," "artillery."

(*b*) Show him common objects—a knife, a pen, a matchbox, etc.—and ask him to name them; or, if he is dumb, to indicate with his fingers the number of syllables in the name of each. If he is unable to fulfil these tests, he has evidently got some forgetfulness of words (**amnesia verbalis**). Sometimes the patient has a general idea of the word he wants to use, but forgets exactly how to pronounce it. He omits some syllables, or substitutes others for them, so that the listener may hardly be able to make out what word it is he wishes to use. This has *been termed* by Wyllie "*articulative amnesia.*"

(*c*) If he makes mistakes in his use of words, calling the knife a pen, or *vice versâ*, he is suffering from **paraphasia.** In that case, one should note whether or not the patient shows that he is aware of his error by trying to correct himself, or whether he goes on talking gibberish.

3. *How is it repeated or echoed?*—Ask him to repeat words after you. If he is word-deaf, try to make clear your request by the aid of pantomime, repeating the word or phrase over and over again. If he is able to repeat what you say, endeavour to find out whether or not he understands what he is saying.

II. **Written speech.**

1. *How is it received or interpreted?*—Ascertain whether or not his sight is good. If so, write on a piece of paper such questions or commands as, How old are you? Put out your tongue; etc. If he does not respond satisfactorily, there is evidently some word-blindness present—*i.e.* the patient has **visual aphasia.**

2. *How is it produced?*—Ask him to write his name. (This can often be done when all other power of writing is lost.) If he is able to do so, ask him some simple question—*e.g.* How many do two and two make?—and get him to write a reply. If he has word-deafness, put your question in writing. If his right hand is paralysed, make him write or print with his left. If he writes pretty well, get him to write an account of his illness, and note whether he makes use of the wrong word at times (paragraphia), or whether there is repeated use of any particular word.

3. *Can he write to dictation or copy?*—Try, using some simple book. If he succeeds, endeavour to ascertain whether or not he understands the meaning of what he writes.

III. Phenomena associated with speech.

1. *Does he understand pantomime?* Does he nod his head for "yes," shake it for "no," and can he indicate numbers with his fingers? Loss of gesture language is termed **amimia.** Mistakes in the use of gestures—*e.g.* nodding for "no," or shaking the head for "yes"—is termed **paramimia.**

2. *Does he understand symbols—e.g.* numerals? Thus, one may write down—

$$\begin{array}{ccc} 2 & 2 & 2 \\ 2 & 2 & 2 \\ \hline 4 & 5 & 6 \end{array}$$

and ask him to point out which is right. If he can read music, test him with musical notes.

3. *Can he recognise common objects?*—Place beside him a pencil, a coin, and a match. Ask him to strike a light, or to write something down. If he is unable to select the proper article for the purpose, he is suffering from **mind-blindness.** Inability to recognise his friends is another proof of the same condition.

SECTION III.—CRANIAL NERVE FUNCTIONS.

In this section we propose to give a brief *résumé* of the essential points in the anatomy of each cranial nerve, to indicate its functions, and, in some cases, the chief symptoms which result from its paralysis, and then to describe the method in which one investigates the state of the nerve at the bedside.

First or olfactory nerve.

Anatomy.—The nerve fibres which arise from the olfactory bulb are distributed to the Schneiderian membrane, at the *upper part of* the nasal fossæ. The cortical centre for smell is believed to lie in the uncinate gyrus. The exact course of the fibres between the cortex and the bulb is unknown, but it is probable that some of them do not decussate.

Test.—Have three small bottles containing some oil of cloves; some oil of peppermint, and some tincture of asafœtida. Apply these to each nostril separately, and ask the patient if he recognises them. In testing, avoid the use of such irritating substances as ammonia, for these act, partly at least, through the fifth nerve. The sense of smell may be abolished. This is known as **anosmia.** Before concluding that the nerve is at fault, take care to exclude local changes in the nose itself—*e.g.* catarrh. **Parosmia** is the name applied to a condition in which the sense of smell is perverted, so that, for instance, offensive substances seem to have a pleasant odour, and *vice versâ.*

Inquire also regarding **hallucinations of smell.** These sometimes constitute the aura of an epileptic fit.

Second or optic nerve.

Anatomy.—From the retina, which is the end-organ of the sense of sight, the fibres of the optic nerve pass back to the optic chiasma. Here the fibres from the inner half of each retina decussate, whilst those from the outer half remain on the same side. Each optic tract, therefore, consists of fibres from the other half of the retina on the same side and the inner half of the retina on the opposite side. Each tract passes back to the corpora quadrigemina, thence the fibres pass to the external geniculate body of the same side, then reach the posterior limb of the internal capsule, on leaving which they spread out in the optic radiation to the cortex around the calcarine fissure. This, therefore, constitutes the primary visual centre, and represents the opposite half of the field of vision, the left half of the field of vision being represented in the cortex of the right hemisphere, and *vice versâ.* From the primary visual centre fibres pass to the angular and supramarginal gyri of the same side, and these constitute a higher visual centre. In this higher centre word images would appear to be stored, and in it also both eyes would seem to be represented, but the field of the opposite eye to a much greater extent than *that* on the same sdie (Fig. 122).

Test.—In testing the optic nerve, one has to investigate three functions: (1) Acuity of vision; (2) extent of field of vision; (3) colour sense. We shall consider the methods of testing these *seriatim*.

Certain preliminaries must always be attended to. One of these is to see that any error of refraction in the patient's eyes is first corrected, and that there is no opacity of his media; another is to take care to examine each eye separately.

1. **Acuity of vision.**—If this be very much diminished, it may be doubtful whether the patient is able to tell light from darkness. To investigate this, place the patient in a darkened room opposite to a lamp, alternately cover and uncover his eye, or, what is perhaps a better plan, concentrate the light upon his eye by means of a mirror or lens, and ask him to say when it is light and when it is dark.

In lesser degrees of impairment, ask the patient to count fingers. This is done by placing him with his back to the light while the observer, standing facing the patient, holds up a varying number of fingers of one hand, and asks the patient to say how many there are. The test should be applied at varying distances.

For the detection of slight degrees of impairment of visual acuity Snellen's types will be found useful. These consist of letters of different sizes, each of which should be capable of being read at a definite distance—the largest at 60 metres, the smallest at 6. In using the types, the patient is placed with his back to the light, while the types are placed level with the eye at a distance of 6 metres (about 20 feet). He is then asked to read the letters from above downwards. For the purpose of recording the result, the following symbols are employed:—

V = visual acuity.

d = distance of eye from type (*i.e.* 6 metres).

D = distance at which type should be capable of being read.

Suppose that at 6 metres the patient is able to read the smallest type—that is to say, that which should be readable at 6 metres off. Then his visual acuity (V) $= \frac{d\ (i.e.\ 6\ \text{metres})}{D\ (i.e.\ 6\ \text{metres})}$ or normal.

But if at that distance he can only read the largest size of type—that which one should be able to read at 60 metres—then V $= \frac{6}{60}$.

The term **amblyopia** (literally "blunt-eyedness") is often used to mean defective vision without any visible change in the fundus oculi, or with signs of mere optic atrophy. By **crossed amblyopia** one means dimness of vision in one eye, there being a lesion in the opposite half of the brain. It occurs, for instance, not unfrequently, in hysterical hemianæsthesia on the same side as the loss of sensation. The term **amaurosis** (literally "darkness") used to signify complete blindness with a similar absence of visible change.* It must be confessed, however, that these terms are used very vaguely, and as far as possible they should be avoided.

2. **Extent of field of vision.**—For ordinary clinical purposes the extent of the field of vision can be tested with sufficient accuracy in the following way:—

Seat yourself opposite to the patient and at a distance of about half a yard from him. If his right eye is to be tested, ask him to place his hand upon his left, and to look steadily at your own *left* eye. Look steadily yourself at the patient's right eye, your own right being closed, and hold up your left hand in a plane midway between his face and your own, and at first at almost full arm's length off. Keep moving the fingers of the hand, and bring it nearer until you can just yourself "with the tail of your eye" catch the movement of the fingers. Then ask the patient whether he sees them, telling him meanwhile to be sure not to take his own eye off yours. If he fails to see the fingers, keep bringing the hand nearer until he sees them. Test the field in this fashion in every direction—upwards, downwards, to right, and to left, using the extent of your own field always for purpose of comparison.

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* Amaurosis has been aptly defined as a condition in which the patient sees nothing, nor the doctor either.

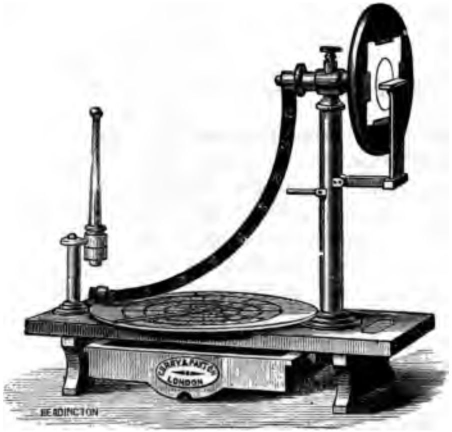

Fig. 123.—Priestley Smith's perimeter.

For more accurate delimitation of the field of vision one must make use of a **perimeter.** Priestley Smith's is a good form, and is shown in Fig. 123. It *is used as* follows:—

1. The patient rests his cheek against the wooden pillar so that the eye is about an inch and a half above the knob and vertically over it. The height of the instrument is regulated by movable blocks.

2. The quadrant, which is a flat strip of metal engraved upon its two sides, is rotated by a wooden hand-wheel attached to the axis; it is balanced by a weight upon the hand-wheel, so that it will stand in any position without being fixed.

3. The test-object is a square of paper gummed upon a light vulcanite wand which the operator holds in the left hand. With the right hand he rotates the hand-wheel and pricks the chart.

4. The chart is placed upon the hinder surface of the hand-wheel, and rotates with it. There is a mark on the hand-wheel to show which way the chart is to be placed. This mark is brought to the top, and the chart is then slipped in from above downwards and in the upright position.

5. Immediately behind the hand-wheel is fixed a horizontal scale, the divisions of which correspond with the circles on the chart. As the quadrant rotates the chart rotates with it, and in whatever position the quadrant stands, the corresponding meridian of the chart stands against the scale. This arrangement enables the operator to prick off his observations with the greatest ease, and has the further advantage that the chart is constantly under inspection, so that any portion of the field can be immediately brought under examination at any time.

6. The charts are of two kinds, A and B. The A charts correspond to the entire field and are divided by circles from 0° to 90°, the limits of the average normal field being shown by a dotted line. The B charts are for mapping the central part of the field on a larger scale, and are divided from 0° to 45°. The scale of the perimeter is graduated accordingly on its two sides; the A side is to be used with the A charts, the B side with the B charts.

7. There are many cases in which it is better to sweep the field, or parts of it, in circles rather than in meridians, *e.g.* hemiopic and sector-like defects in which the boundary line of the field runs in a meridianal direction. In cases of this kind the test-objects may be placed in the clip upon the quadrant, and carried round the field in successive circles.

McHardy's instrument is shown in Fig. 124. The following points should be attended to in using it:—

The chin rest (*E*) is adjustable to suit each patient, the level of the eye is then gauged by the button (I) on the sliding stem (*H*); next, "zero" is raised, and fixed by the milled head (*b*) to the level of eye, by aid of scales on *a* and *K*. The biting fixation attachment (*L*, *M*) is detachable; it is only to be put in place when required in mapping the "field of fixation," as distinguished from the "field of vision." Registration is accomplished by simply pressing the chart box (*e*) into an upright position. The quadrant (*hh*) rotates smoothly, and remains rigid in any position. The large vulcanite disc (*n*) readily slides into position, so as to screen the operator's cuff while he turns the driving milled head (*j*), which simultaneously manipulates the travelling carriage (*i*) and the pointer

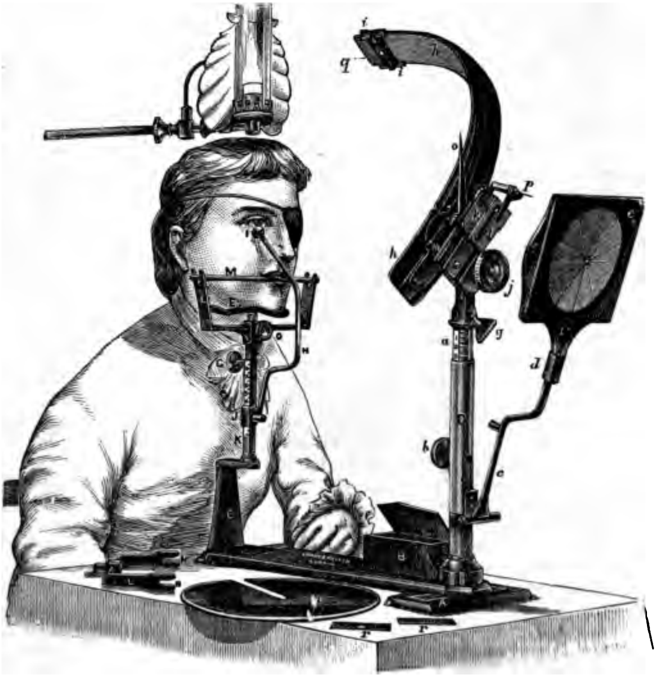

Fig. 124.– McHardy's perimeter (*for details, see text*).

(*p*) of the automatic registration. The skeleton charts are printed red, and correspond to the entire field, and are divided by circles from 0° to 90°, the limits of the average normal field being shown by a faint line. Any portion of the field can be immediately brought under examination, while the records are not liable to be biassed as when under inspection. The field or parts of it may be as readily swept in circles as in meridians, in those cases in which the former method is preferable; *e.g.* for hemiopic and sector-like defects.

The test-objects, circular carriers of coloured paper, are placed in the carriage (*i*) behind various diaphragms (*rr*) with apertures of given diameter. Degrees and half-degrees are marked on the back of both edges of the quadrant, so that the instrument will determine the angle of squint or the angle *a* accurately.

Ascher's perimeter, which is constructed mainly of celluloid, is a light and portable form for private use.

Changes in the field of vision.—It may be contracted all round its periphery. This is spoken of as "*concentric diminution*" of the field of vision. It occurs in hysteria, optic atrophy, and various affections of the retina.

Sometimes the loss of vision is confined to the centre of the field. This is spoken of as a *central scotoma* or as *central amblyopia.* It is frequently due to toxic causes—*e.g.* excess in tobacco or alcohol, and is then generally bilateral. Sometimes it is due to local disease of the choroid or of the retina in the neighbourhood of the macula. In that case it may affect only one eye.

The term **hemianopsia** (also written hemianopia and hemiopia) means loss of sight in one-half of the field of vision in both eyes from causes other than disease in the retina. Right lateral hemianopsia means abolition of the right half of the field of vision; left lateral hemianopsia, abolition of the left half. These forms of hemianopsia are also spoken of as "*homonymous.*"

"*Superior*" and "*inferior*" hemianopsia mean loss of the upper and lower halves of the visual field respectively. They are rarer than the lateral variety, and are sometimes spoken of as "*altitudinal*" *hemianopsia.*

Temporal hemianopsia means loss of vision in the temporal or outer halves of both fields, and is due, therefore, to loss of visual power in the nasal half of each retina. It can only be produced by a lesion either just before or just behind the optic chiasma, involving those fibres of the optic nerves which have decussated, and is accordingly very rare.

Nasal hemianopsia signifies a loss of the nasal or inner half of each field, and indicates a diminution of visual power in the temporal half of each retina. It can only be produced by a bilateral lesion confined to the uncrossed optic fibres on each side of the chiasma. It only occurs with excessive rarity.

Temporal and nasal hemianopsia are sometimes spoken of as *heteronymous*, in contradistinction to the *homonymous* variety.

3. **Colour sense.**—This is tested by means of Holmgren's wools. Throw all the skeins together on a table in good daylight, keeping the test skein separate. Explain to the patient that he is to *match* the colour, not to name it, and that he is to select all those skeins which are *like* it, whether they are of a darker or lighter shade or not. Show him first a pure pale-green skein, and ask him to match it. If he does so correctly, his colour vision is normal. If, on the other hand, he selects one of the "confusion colours" (grey, straw colour, etc.), he is to be regarded as colour-blind.

Total colour-blindness is rare. Red-green blindness is the commonest form. Yellow-blue blindness is not nearly so common. If the patient is totally colour-blind he confuses with the test skein all those of equal brightness, no matter what their tint may be. If red-green blindness is suspected, show him a purple skein, and he will select blue as a match for it—indicating that he fails to see the red element in the purple. If he be blue-blind, he will select red or orange.

Colour field.—In a normal eye the field for blue is largest, then comes yellow, then red, and lastly green. Concentric diminution of the colour field occurs in hysterical amblyopia. In some toxic conditions, especially tobacco poisoning, one finds a central scotoma for colour. Its existence can easily be determined by placing the patient with his back to the light, and then holding up about 2 feet in front of him a square of black pasteboard in the centre of which is a small white spot. Ask him to look steadily at the white spot, and while he does so suddenly place about 2 inches to the outer side of the spot a black strip of cardboard, near the end of which a red or green wafer has been fixed. If a central colour scotoma is present, the patient will not see the red or green spot whilst he is looking at the white one. If, on the other hand, the coloured spot be placed to the inner side of the white spot, he has usually no difficulty in seeing it. Colour-blindness, of course, interferes with the use of the test in some cases.

The exact extent of the field for each colour is best tested by means of the perimeter.

Subjective visual sensations may be present. Amongst the commonest of these for which one may have to inquire is the occurrence of what are known as *muscæ volitantes*—little specks or motes seen floating before the eyes, especially on looking at a white surface or up to the sky. They are not infrequent in anæmic and debilitated persons. In migraine peculiar zigzag lines, known as "fortification figures," are often seen at the beginning of the attack, and in the investigation of such a case should always be inquired for. The term *teichopsia* is also applied to the condition. Hallucinations of sight occur in some cases, notably in delirium tremens; they may also form part of the aura in epilepsy.

Third, fourth, and sixth nerves.—It is convenient to take these together, as conjointly they serve to innervate the muscles which move the eyeball.

Anatomy.—The fibres of these nerves take their origin from a series of nuclei which begin in the floor of the aqueduct of Sylvius below the anterior corpora quadrigemina, and extend down as far as the eminentia teres in the floor of the fourth ventricle. The nucleus for the third nerve is highest up. Its most anterior cells supply the ciliary muscle and iris, those for the ocular muscles being farther back. Behind that comes the nucleus of the fourth, and most posteriorly of all that of the sixth. The third nerve emerges on the inner aspect of the crus, and is therefore apt to be involved in lesions implicating that part of the brain.

The fourth pair emerge on the anterior part of the roof of the fourth ventricle. They are peculiar in that they are the only cranial nerves which decussate between their nuclei and their point of emergence.

The sixth emerges between the medulla and pons, and runs forward beneath the latter for a considerable distance before leaving the skull. This long course renders it particularly liable to the effects of pressure.

Functions.—The sixth nerve supplies the external rectus, the fourth supplies the superior oblique. All the other ocular muscles, along with the sphincter pupillæ, the muscle of accommodation, and the levator palpebræ superioris, are supplied by the third.

Symptoms of paralysis.—*Sixth nerve.* Inability to move the eye outwards and diplopia on looking in that direction. Possibly internal squint. In nuclear lesions there is also loss of the power of conjugate deviation in the direction of the affected muscle.

Fourth nerve.—Impaired power of downward *movement; possibly* upward and inward squint; and *diplopia on looking* down.

Third nerve.—Ptosis; the eye can only be moved outwards and a little downwards and inwards; loss of accommodation; pupil of medium size and unable to contract; loss of power of accommodation.

Paralyses of the third nerve are not unfrequently partial—only one or a few of these functions being lost.

Thus the levator palpebræ superioris is often alone affected, producing ptosis, while the other muscles retain their normal power. In order to estimate the degree of **ptosis,** one must eliminate the action of the occipito-frontalis. This is done by pushing down upon the latter muscle so that the eyebrows are kept level, and then asking the patient to look up. The amount to which the lids are raised indicates the strength of the levator.

Any **retraction of the upper lid,** from over-action of the levator, is to be noted by observing the relation of the head of the lid to the upper margin of the cornea when the patient is looking straight forward.

How to test these nerves.—As will be gathered from the above *résumé*, the signs of a lesion involving any of these nerves may be: 1, The presence of a squint; 2, defective power of movement of the eye; 3, the presence of diplopia. Of these signs the last is really the most trustworthy of all, for paralysis of the muscles supplied by the nerve may be so slight as to lead to no appreciable squint and to no visible defect in mobility.

We shall consider the question of squint first.

1. By **squint** or **strabismus** is meant a want of parallelism in the two visual axes. It may be due either to paralysis of a muscle or to over-action—*i.e.* spasm—of its opponent. The former constitutes **paralytic strabismus,** the other brings about what is called "**concomitant**" (or *spasmodic*) **strabismus.** The first point, therefore, to be decided about

any squint is this—Is it paralytic or is it concomitant? The chief points of distinction between the two are these:—

(*a*) Spasmodic squint is always present; on asking the patient to look straight forward, which is the position of rest of all the ocular muscles, the squint is seen at once. Paralytic strabismus, on the other hand, may only be visible when the patient happens to look in a direction requiring the action of the paralysed muscle.

(*b*) In spasmodic strabismus the affected eye follows the sound eye equally in all its movements. It is for this reason that it is termed "concomitant." The visual axes are not parallel even in the position of rest, and the defect of parallelism remains the same in whatever direction the patient turns his eyes. In paralytic squint the visual axes may appear parallel in the position of rest, but even if they do not, the want of parallelism becomes more and more evident the farther the patient tries to turn his eyes in the direction of action of the paralysed muscle.

(*c*) Diplopia is usually present in paralytic squints, absent in those which are spasmodic.

The commonest form of concomitant squint is the internal strabismus, which occurs in children, and is associated with hypermetropia.

2. **Defective power of movement of the eye.**—In order to elicit this symptom, place the patient with his face to the light; stand in front of him, and, holding up one finger, ask him to follow its movements with his eyes. It will easily be noticed whether or not a squint is brought out in either eye when he attempts to do so. The examination may be repeated in each eye separately, its mobility being tested in each direction.

The power of convergence of the eyes should always be tested in addition to the mobility of each eye separately. In order to do this, hold up your finger directly in front of the patient at a distance of about 18 in. from the tip of his nose. Tell him to keep looking at the finger, which is then gradually brought nearer to the nose. Note to what extent convergence occurs, and whether it is well maintained —*i.e.* whether the eyes remain directed towards each other, or whether they diverge again after their first convergence.

In some cases the defective mobility is so slight as to elude detection. This is especially apt to be the case in paralysis of the oblique muscles. In such a case one falls back upon the diplopia as an indication of the affected muscle.

3. **Diplopia.**—In order to elicit this symptom, hold your finger straight up in front of the patient, and ask him how many there are. Then repeat the question with the finger held at each side of the visual field, then high up, and then low down. Make sure that the patient's head is not moved during the investigation. If in each position he sees one finger only there is no diplopia. If at any part of the field two fingers are seen—one distinct, the other somewhat hazy—one may be sure that diplopia is present.

In that case one has next to ascertain (1) which is the affected eye; (2) which is the affected muscle in that eye.

In order to determine these points, proceed as follows:—

Place over one of the patient's eyes a red glass—preferably over the stronger eye, if he has better vision in one than the other. Then hold up in front of him a tall lighted candle. Move it about until he sees two candles—a red and a yellow. One of these is the *true* image—*i.e.* that of the sound eye; the other is the false image—*i.e.* that of the affected eye.

Which is which? In order to answer this question the following rule is given :—

The affected eye is that in the direction of the image of which the diplopia increases.

The application of this rule will be made plain by an example. Suppose the red glass is opposite the patient's left eye, and the patient says that the red image is to the right of the yellow. On moving the candle farther to the right the distance between the images increases—*i.e.* the diplopia becomes greater—that is to say, it becomes greater on moving the candle in the direction of the red image. But that is the image belonging to the left eye; therefore, applying the rule, the left eye is the one that is affected.

The affected eye having been thus discovered, one wishes to know which is the affected muscle. To help one in this a second rule has been given :—

The paralysed muscle is that which would have turned the eye in the position and direction of the false image.

In the above example one found that the left eye was the one affected, and that its image was to the right of the true image. Applying the above rule, one asks, Which muscle moves the left eye to the right? and the reply is, The internal rectus. The left internal rectus, therefore, is the affected muscle.

When, as in this case, the false image is on the opposite side to the affected eye, the diplopia is said to be "*crossed*"; when the false image is on the same side as the affected eye, the diplopia is said to be "*direct.*" Paralysis of an internal rectus always produces a crossed diplopia; paralysis of an external rectus a direct diplopia.

The detection of the affected muscle in cases of **vertical diplopia** is somewhat more difficult than in cases where the diplopia is lateral. The same rules, *however*, apply. Suppose, again, that the red glass is over the left eye, and that the two images are seen, one above the other, the yellow being higher up. On looking upwards the distance between the images becomes greater; therefore, according to the first rule, the right eye is the one affected. *Which is the paralysed muscle?* In order to apply the second rule, one must remember the action of each muscle, and, from the position of the false image as described by the patient, deduce which muscle it is which would have turned the eye in that position and direction. This will be the muscle affected. Werner's diagrams (Figs. 125, 126) facilitate the recollection of this greatly. The continuous lines on the diagrams represent the positions of the true images, the dotted lines those of the false images. Thus in paralysis of the left inferior rectus the false image is at a lower level than the true, it is to the right side of the true image (crossed diplopia), and the upper end is inclined towards the true image (Fig. 125). In paralysis of the left inferior oblique, the false image is higher up than the true image and to its left side (direct diplopia), and its upper end is inclined away from the true image (Fig. 126).

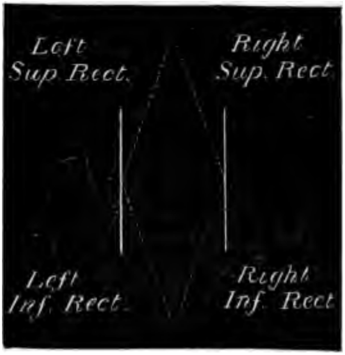

Fig. 125.—Werner's diagram.

To return, then, to our supposed case. One had proved that the right eye was the one affected, and that its image was higher up than the true image. The paralysis must therefore affect either the right superior rectus or the right inferior oblique. Ask the patient whether the upper end of the false image is inclined towards or away from the true, and whether it is to the right or the left of the latter. Supposing he says that the false image (yellow candle) is to the left of the true, and with its upper end inclined away from the latter. Then one knows at once, by referring to the diagram (Fig. 125), that the right superior rectus is the muscle affected.

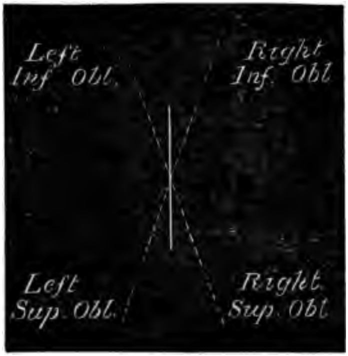

Fig. 126.--Werner's diagram.

If, in the above example, the patient is unable to state clearly whether the false image is to the right or left of the true, and what is the exact nature of its inclination, one can have recourse to another method. Ask in what direction the difference in height of the two images increases. If towards the temple, the rectus is the affected muscle; if towards the nose, it is the oblique.

In applying the above tests it is more convenient to move the patient's head in order to change the direction of his eyes than to move the candle. Thus if one turns the head to the left and asks him to look at the candle the eyes are turned to the right. Thus moving the head to the left comes to the same thing as moving the candle to the right, and *vice versâ.* To lower the eyes, hold the head back; to elevate the eyes, depress the chin. These movements are *effected by* the observer, who places himself at the patient's side, the lighted candle being placed on a table some distance off.

The **position of the patient's head** is also of considerable help in detecting the paralysed muscle. He tries, by turning his head, to give to the affected eye the position in which it should be placed by the paralysed muscle. Thus, if the face is turned to the left, it indicates a paralysis of the left external rectus or right internal rectus, and *vice versâ*. If it be directed upwards, an elevator is paralysed; if downwards, a depressor. If the head is inclined to the left shoulder, it indicates paralysis of the superior rectus or oblique of the right eye, or of the inferior rectus or oblique of the left eye. If the inclination of the head is towards the right shoulder, it indicates paralysis of the superior rectus or oblique of the left eye, or of the inferior rectus or oblique of the right eye.

Abnormal movements of the eye.

Involuntary clonic contractions of the muscles of the eyeball not unfrequently occur. The movements are usually symmetrical, occurring equally in both eyes. To these movements the term **nystagmus** is applied. If the external or internal recti are affected, *lateral* nystagmus results. *Vertical* nystagmus is due to an affection of the superior or inferior recti, and *rotary* nystagmus to an involvement of the oblique muscles. Lateral nystagmus is the commonest variety.

In examining for nystagmus, ask the patient to look straight in front of him, and observe whether the eyes remain steady. Then ask him to look to his extreme right, then to the left, and then upwards and downwards. Nystagmic movements are frequently only to be observed when the eyes are turned as far as possible in one of these directions. Such an occurrence is indicative of paresis of the muscles which turn the eyes in that particular direction.

If both eyes are kept persistently turned in one direction, the condition is spoken of as **conjugate deviation** of the eyes. It is usually either to the right or to the left. Conjugate deviation of the eyes may be brought about either by a lesion which produces paralysis or by one which causes irritation or spasm. In the former case the eyes (and usually, also, the head) are turned towards the side of the lesion, provided the latter be in the cerebral hemisphere. The patient, in fact, is said "to look towards his lesion." An irritative lesion in a similar situation causes the deviation to be towards the healthy side. If, however, the lesion have its seat in the pons, these rules are just reversed, the deviation being towards the sound side in a paralytic lesion, and towards the affected side in one which is irritative.

Examination of the pupils.—This important part of the investigation of a nervous case may be conveniently considered at this stage. The following points must be noted about the pupils in every case:—

1. **Size.**—Compare the size of the two pupils, first in a bright light and then in a dim light. Note whether the pupils are large or small, and whether any irregularity is present. It must be remembered that the size of the pupil in health is subject to great variations. As a rule, the pupils are larger in dark eyes than in light. A much dilated pupil is often a sign of nervous exhaustion or instability. Slight inequality of the pupils may also be present in perfectly healthy subjects. We are inclined to think that in such cases the left pupil is usually the larger.

If one pupil is larger than the other, the question arises: Which is the normal? This question is not always very easily answered, but, as a rule, the pupil which exhibits the least mobility is to be regarded as *the abnormal* one.

2. **Shape.**—Note whether the pupil is circular in outline, as it should be, or whether its contour is irregular. Such irregularities may be due to adhesion of the iris to the lens (*see* p. 502). Irregularity in shape of the pupil is often an early symptom in general paralysis of the insane.

3. **Mobility.**

(*a*) **Reaction to light.**—This is a reflex action. The stimulus is produced by the action of light on the retina, and travels along the optic nerve and optic tract to the corpora quadrigemina, and thence by communicating fibres (Meynert's fibres) to the centre for the third nerve. The motor impulse passes from that centre by means of the fibres of the third nerve to the sphincter pupillæ muscle (Fig. 127).

Test.—Examine each eye separately. Place the patient opposite a bright light, be sure his accommodation is relaxed, and cover the eye with the hand. Leave it covered for about half a minute, then withdraw the hand and watch the pupil. It should contract almost immediately, then dilate again a little, and, after undergoing slight oscillations, settle down to its normal size.

The test may also be carried out by concentrating light upon the pupil by means of a mirror or lens, just as one does in testing the light perception.*

Owing to the decussation of some of the fibres of the optic nerves at the chiasma, light acting upon one eye affects the centre for pupil contraction of the other eye as well as that on its own side. It is probable that fibres pass directly between the centres for the two third nerves which aid in bringing about this result. As a consequence, one finds that if light be shut off from one eye both pupils dilate, and if bright light be made to enter one eye both pupils contract. This is known as the **consensual reaction** of the pupils. It should be tested by keeping one eye in the shade while light is thrown into the other. The effect on the pupil of the shaded eye is then observed.

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* A convenient method is to throw the light on the pupil by the mirror of an ophthalmoscope with a + 8 lens behind. Looking through the lens one gets a magnified view of the pupil, and small *changes in it* can be more easily observed.

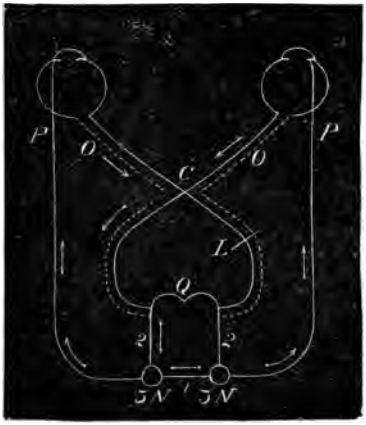

Fig. 127.—Diagram showing the connections of the centres for contraction of the pupils. (*Swanzy.*)
3 N, 3 N, centre; 1, connection between nuclei of 3rd nerves; 2, Meynert's fibres; Q, corpora quadrigemina; C, chiasma; O, optic nerve; P, pupil contracting fibres of 3rd; L, seat of lesion; arrows show path of impulse in lesion of right tract at L.

Lesions of the optic nerve or optic tract interfere with this reflex.

Wernicke's hemiopic pupil reaction may be mentioned here. Hemianopia, as we have seen, may be due to a lesion of the optic tract between the chiasma and the corpora quadrigemina, or it may be produced by destruction of the optic fibres between the corpora quadrigemina and the occipital cortex, or it may be due to lesion in the cortical visual centres themselves.

If the lesion be in front of the corpora quadrigemina—*i.e.* in front of the pupil centre—the light reflex is lost; whereas, if it be at any point behind that, the contraction of the pupil to light is retained. It is upon this fact that Wernicke's reaction is based.

In carrying out the test the light must, of course, be concentrated on the blind halves of the retinæ. Proceed as follows: Place the patient in a dark room with a light beside his head. Hold a large plane mirror in the left hand, and by means of it illuminate both pupils and observe their size. Then take an ordinary opthalmoscopic mirror in the right hand and direct a strong beam of light on to the blind side of the retinæ. If the lesion be in front of the corpora quadrigemina no contraction of the pupils should result; if behind that, they become smaller.

(*b*) **Reaction to accommodation.**

As is well known, the pupils become smaller on accommodating for a near object. It is really more correct to speak of reaction to *convergence*, as it is found that it is the convergence of the eyes, not the mere effort of accommodation, which causes the pupil to become smaller.

Test.—Hold up one finger close to the patient's nose. Ask him to look away at a distant object. Then suddenly tell him to look at your finger. As the eyes converge to accomplish this the pupils should become decidedly smaller.

If the patient is unable to see, the test may still be carried out by getting him to hold up his own finger about a foot in front of his face and then asking him to direct his eyes to it.

If the pupil reacts to accommodation but not to light, it is probable that there is a lesion either of the optic or of Meynert's fibres. If it reacts neither to light nor to accommodation, it is probable that there is a lesion either of the pupil centre or of the fibres of the third nerve.

Argyll-Robertson pupil.—This is the term applied to the condition of pupil usually observed in locomotor ataxy, but also found sometimes in general paralysis and other degenerative diseases of the nervous system, especially syphilitic. It reacts to accommodation, but not to light. It is probably due to a lesion in Meynert's fibres (Fig. 127)—*i.e.* the fibres of communication between the corpora quadrigemina and the pupil centre. Sometimes the reaction to light is not entirely absent, but takes place in a very sluggish fashion.

(*c*) **Cilio-spinal reflex.**—Dilatation of the pupil can often be observed to follow irritation of the skin of the neck either by pinching or by the action of a Faradic current. It is due to stimulation of the pupil-dilating fibres in the cervical sympathetic (p. 456), and is abolished in lesions of that nerve.

Abnormal movements of the pupil.

The term "**hippus**" is applied to the alternate contraction and dilatation of the pupil, which can sometimes be observed going on rhythmically (*see* p. 502).

Fifth nerve.

Anatomy.—**1.** The **sensory root** takes origin in a large nucleus in the pons, situated in the floor of the fourth ventricle and lying external to the motor nucleus, and partly also from the "ascending" root, which begins as low down as the second cervical segment. It emerges at the side of the pons, and, immediately beyond the Gasserian ganglion, separates into its three divisions.

The **first, or ophthalmic, division** supplies the eyeball, conjunctiva (except that of the lower lid) and lachrymal gland, the mesial part of the skin of the nose as far as the tip, the upper eyelids, the forehead, and the scalp as far as the vertex.

Paralysis of this division results in loss of sensibility in the area of skin and mucous membrane supplied, and in trophic changes in the eyeball (if the lesions involve the Gasserian ganglion). The conjunctival reflex is abolished.

The **second or superior maxillary division** supplies the cheek, the front of the temple, the lower eyelid and its conjunctiva, the side of the nose, the upper lip, the upper teeth, the lining membrane of the nose, the upper part of the pharynx, the roof of the mouth, the soft palate, and the tonsils.

Paralysis of it leads to abolition of sensibility in the above area, and loss of the palate reflex.

The **third or inferior maxillary division** is joined by the motor root. It supplies sensation to the lower part of the face, the lower lip, the side of the head, the ear, the tongue, and the lower teeth. It supplies also the salivary glands and, through the motor division, the muscles of mastication, the tensor tympani, and also, perhaps, the tensor palati, although many believe that this muscle is innervated by the spinal accessory.

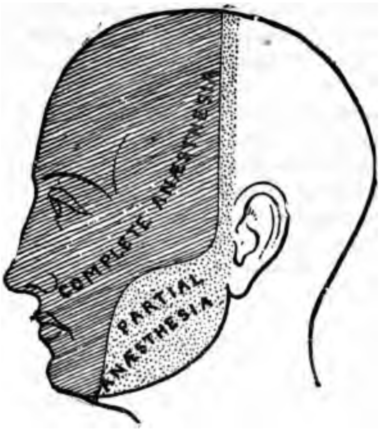

Fig. 128.—Distribution of anæsthesia in complete paralysis of the fifth nerve.

2. Motor root. This takes origin in a small nucleus lying internally to the chief sensory nucleus, and partly also from the descending root, which arises in nerve cells scattered around the aqueduct of Sylvius. It emerges at the side of the pons, just in front of the sensory division, passes underneath the Gasserian ganglion, and joins the inferior maxillary division, to which it gives its motor fibres.

Paralysis of the whole fifth nerve leads to loss of sensation in the areas of skin and mucous membrane above mentioned, and to defective power of chewing. (Fig. 128.) Trophic lesions may be present and the salivary, buccal, and lachrymal secretions much diminished, and the sense of taste abolished.

One curious result of the sensory paralysis is that the patient, when drinking, imagines that the cup is broken, as he only feels it on one side of his mouth.

How to test the fifth nerve.

1. **Motor functions.**—Ask the patient to clench his teeth while the observer keeps his hands on the temporal and then on the masseter muscles. These should stand out with equal prominence on each side. If there be paralysis on one side, the muscles on that side will fail to become prominent. On opening the mouth the jaw deviates towards the paralysed side, being pushed over by the healthy external pterygoid muscles. The condition of the tensor tympani muscle cannot be satisfactorily examined except by noting whether there is any difficulty in hearing notes of a particular pitch—*i.e.* a diminution in the "field of hearing."

2. **Sensory functions.**—The common sensibility of the area supplied is tested in the usual way (p. 469).

Taste.—In suspected lesions of the fifth nerve the sense of taste should always be examined. It seems probable that all the taste fibres eventually reach the brain in the fifth nerve. The taste fibres from the anterior two-thirds of the tongue pass from the lingual nerve to the chorda tympani, thence to the facial, thence by the great superficial petrosal to Meckel's ganglion, and thence to the second division of the fifth.

The taste fibres from the posterior third of the tongue enter the glossopharyngeal nerve, leave it in the nerve of Jacobson, reach the tympanic plexus, and thence by the small superficial petrosal and otic ganglia arrive at the third division of the fifth.

Once the taste fibres have entered the brain their exact course becomes doubtful. There is no doubt that they reach the posterior part of the internal capsule, and by that time they have already decussated.

It is probable that they end in the tip of the temporo-sphenoidal lobe.

How to test the sense of taste.—Have some sugar, some quinine, and some salt, all in powder. Ask the patient to put out his tongue and to keep it out until the conclusion of the test. Place some sugar on the tongue, rub it gently in, and ask him, "Is that salt?" If taste is normal he will shake his head. In this way all the substances are tried, first on the anterior part of the tongue and then at the back. A weak galvanic current is also a useful test. It should produce a sort of metallic taste.

Loss of taste may, of course, arise from lesions of the taste fibres in any part of their course above stated.

In addition to loss of taste, one should always ask the patient whether he has any abnormal taste sensations.

Seventh nerve.

Anatomy.—The course of the fibres of this nerve from the cortical centre to the nucleus has already been described p. 397). The nucleus is situated in the pons external to that of the sixth nerve. The fibres which are destined to supply the orbicularis palpebrarum, however, possibly proceed from the nucleus of the third, and those for the obicularis oris from that of the twelfth nerve. On leaving the nucleus the fibres wind round the nucleus of the sixth, and finally emerge along with the eighth nerve, between the olive and restiform bodies.

The nerve lies in close contact with the eighth, so that a lesion of the one at this part can hardly avoid injuring the other, and enters the internal auditory meatus along with it. During its course in the aqueduct of Fallopius it gives off a branch to the stapedius muscle, and is joined by the chorda tympani, which contains taste fibres from the anterior two-thirds of the tongue. In this part of its course the nerve is exposed to the effects of pressure, owing to its being enclosed in a bony tube. It emerges at a point opposite the junction of the anterior border of the mastoid with the ear, and spreads out on the side of the face to supply its muscles. In this part of its course it seems to be peculiarly liable to the effects of exposure to cold.

Functions.—The seventh is a purely motor nerve. It supplies all the muscles of the face and scalp, except the levator palpebræ superioris. It also supplies the platysma.*

Effects of paralysis.—These are usually at once seen on looking at the patient. The affected side of the face has lost its expression. The naso-labial fold is less pronounced than on the other side. The furrows of the brow are smoothed out, the eye is more widely open than the other, and the mouth is somewhat drawn to the healthy side. The patient is unable to whistle, and food is apt to collect between his teeth and his gums.

How to test the seventh nerve.

1. Ask the patient to shut his eyes as tightly as ever he can. Note that the affected eye is either not closed at all—in which case the eyeball rolls upwards to make up for the failure of the lid to descend—or, if the eye is closed, the eyelashes are not so far rolled in as on the healthy side. Try also forcibly to open the eyes while the patient attempts to keep them closed. If the orbicularis is acting normally, it should be almost impossible to open the eye against the patient's wish. If the muscle be partially paralysed, however, the exertion of very little force may suffice to open it.

The effort made in screwing the eyes tightly shut causes the corners of the mouth to be drawn upwards. In paralysis of the lower part of the face the corner on the affected side is either not drawn up at all, or at all events not so much as on the healthy side.

2. Ask the patient to whistle. He is unable to do so.

3. Ask him to smile or show his upper teeth. The mouth is then drawn to the healthy side.

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* *Paralysis of the* platysma is said to be characteristic *of organic as opposed to* functional lesions.

Signs of paralysis of the facial nerve in different parts of its course.

Paralysis of the seventh nerve produces different symptoms according as the lesion is situated above the nucleus or either at the nucleus or below it. The former constitutes what is known as cerebral or supranuclear facial paralysis, the latter produces peripheral or infranuclear paralysis.

The chief difference between the two forms is that in **supranuclear paralysis** the lower part of the face is chiefly affected ; in **infranuclear paralysis** both the upper and lower parts are equally involved. The probable explanation of this is that the two orbicularis palpebrarum muscles are so often required to act together that each is supplied from both sides of the brain, and consequently a unilateral lesion only partially cuts off the nerve impulses to one side. We have already described what is meant by "crossed paralysis," and the part which the facial plays in it.

Infranuclear facial paralysis may be produced by a lesion of the nucleus itself, of the nerve trunk inside the aqueduct, or of the nerve trunk either after its emergence from the aqueduct or before it has entered it.

A lesion inside the aqueduct—unless it be towards the outer end of the latter—involves the fibres of the chorda tympani, and therefore produces a paralysis of taste sensation in the anterior two-thirds of the tongue. A lesion in any of the other situations produces a typical complete facial paralysis (Bell's paralysis).

A lesion of the nerve before it has entered the aqueduct can be distinguished from a lesion below the stylo-mastoid foramen by the fact that in the former condition the stapedius muscle is paralysed (causing excessive sensitiveness to loud sounds, or "hyperacusis"), while in the latter it escapes. Never omit, therefore, in a case of facial paralysis to inquire regarding the patient's sensitiveness to loud sounds.

The practical deduction from these facts is that in examining a case of facial paralysis one should never omit to investigate the taste sensibility of the anterior part of the tongue.

Lesions of the nucleus or nerve below it will result in atrophy of the facial muscles and the appearance in them of the reaction of degeneration supranuclear lesions do not produce this effect.

Abnormal facial movements.—The muscles supplied by the facial nerve are frequently affected by spasmodic movements. These may involve all the facial muscles or groups of them only. The spasm may be of either the clonic or tonic variety (p. 464). The nature of the movements, if present, their extent and the muscles affected by them, should always be carefully noted.

Eighth Nerve (auditory).

Anatomy.—This nerve consists of two sets of fibres. One set supplies the cochlea, and subserves the function of hearing; the other part supplies the vestibule and semicircular canal, and is the nerve of equilibration. The auditory fibres arise from a nucleus which is mesially placed in the floor of the fourth ventricle. They emerge dorsally to the restiform body, forming the dorsal root of the eighth nerve. The fibres for equilibration take origin in a nucleus placed laterally to the other, and emerge on the ventral aspect of the restiform body forming the ventral root of the eighth nerve. The two roots join, and the fibres run together to the bottom of the internal auditory meatus, where they separate, to be distributed to the different parts of the inner ear.

The cortical centre for hearing is in the first and second temporo-sphenoidal convolutions of the opposite hemisphere. The exact course of the fibres between the nucleus and the cortex is unknown.

The cortical centre for the fibres of equilibration is probubly situated in the cerebellum, but as to that nothing is definitely known.

Test. 1. **Hearing.**—Before testing a patient's power of hearing it is well to exclude the presence of wax in the ear (*see* p. 517). This being disposed of, one can test the hearing power by means of a watch. Stand behind the patient and ask him to shut his eyes. Begin outside the probable range of hearing power and bring the watch gradually nearer the ear, asking the patient to speak whenever he hears the tick. One requires, of course, to know at what distance the tick should be audible by a healthy ear. It is necessary to test each ear separately, one being closed whilst the other is being examined.

If impairment of hearing be detected, one must next try to ascertain whether it is really due to disease of the auditory nerve or merely to some affection of the middle ear. In order to settle this point, the **tuning fork test** may be employed. Strike a tuning fork and place the end of it against the centre of the patient's forehead. If the deafness discovered by the watch is due to an affection of the middle ear, the patient will hear the tuning fork *louder* on that side than on the healthy one. On the other hand, if the deafness is due to disease of the auditory nerve, the tuning fork will only be heard on the healthy side. The test may also be carried out by means of the watch. In affections of the nerve, the watch is not heard even when pressed against the ear; in disease of the middle ear, it is heard even more loudly than when similarly applied to the healthy side. The explanation of these facts is not yet clearly made out, nor are they invariably trustworthy. They hold good, however, for a majority of cases. Other points in favour of the deafness being due to the nerve and not to the middle ear are, (*a*) if the hearing is better in a quiet place, (*b*) if conversation is heard better than the watch, (*c*) if inflation of the middle ear renders the hearing worse.

Abnormal auditory sensations. — The patient may complain of "ringing in the ears," or **tinnitus.** The precise character of the sound varies in different cases. It may be of a humming, buzzing, hammering, or whistling character. The presence or absence of this symptom should always be inquired for, and whether it is constantly present or in what circumstances it comes on.

Hyperæsthesia of the auditory nerve (**hyperacusis**), by which even slight sounds are heard with painful intensity, sometimes occurs, especially in hysteria and in lesions of the facial nerve above or in the aqueduct owing to paralysis of the stapedius muscle.

Hallucinations of sound may also be present, the patient fancying that he hears voices, bells, etc. The presence of these points to an affection of the cerebral cortex.

2. **Equilibration.**—This sense cannot be conveniently tested clinically. Disorders of it produce **vertigo.** The patient will describe this as giddiness or dizziness. In order to constitute true vertigo, external objects should seem to move round him. Ask if this is so, and, if it is, in what direction the objects seem to move. Ask also whether the vertigo ever causes him to fall to the ground.

Vertigo may be due to paralysis of one of the ocular muscles, to an affection of the ear or auditory nerve, to loss of muscular or common sensibility, especially in the lower extremities, or to an affection of the stomach. When a patient complains of vertigo as his chief symptom one should therefore examine carefully for squint, for disease of the outer and middle ear, and for signs of disease of the eighth nerve. One should also investigate the condition of muscular and common sensibility, and inquire for symptoms pointing to gastric disturbance.

The **ninth (glossopharyngeal), tenth (vagus) and eleventh (spinal accessory) nerves.**

Anatomy.—These arise in order from above downwards from an elongated nucleus in the floor of the fourth ventricle. The ninth has also an ascending root, beginning as low down as the fourth cervical segment. They emerge by several roots along the lateral aspect of the medulla, beginning above in the groove between the olive and restiform bodies. The spinal part of the eleventh emerges from the lateral column of the cord, beginning as low as the sixth cervical nerve; it passes up through the foramen magnum to join the medullary (accessory) part, and emerges with it through the jugular foramen. After its emergence the two divisions of it again part company, the medullary or accessory portion joining the vagus.

Functions.

The **ninth** (glossopharyngeal) is sensory for the posterior third of the tongue and for the mucous membrane of the pharynx. It is motor for the middle constrictor of the pharynx and for the stylo-pharyngeus. It contains in part of its course the taste fibres for the posterior part of the tongue (*see* p. 446).

How to test the glossopharyngeal.—The ninth nerve is rarely paralysed alone. Paralysis of it can only be diagnosed by examining its sensory functions. Examine the power of taste in the posterior part of the tongue (p. 447). Loss of it *may* mean paralysis of the trunk of the glossopharyngeal nerve. In root affections of the nerve, taste is not implicated, as the taste fibres enter the brain by the fifth.

Tickle the back of the pharynx, and note if the reflex is present.

The **tenth** (vagus) is motor for the soft palate (with the exception of the tensor palati), pharynx, and larynx. It is also sensory and motor for the respiratory passages, the heart, and (through the sympathetic ganglia) for most of the abdominal viscera.

The fibres for the **soft palate** take origin in the nucleus ambiguus, emerge in the upper roots of the eleventh, reach the pharyngeal plexus, and thence pass to the muscles of the palate and constrictors of pharynx.

The motor fibres for the larynx, the viscero-motor, and the cardio-inhibitory fibres are really derived from the medullary or accessory part of the *spinal accessory*, which, as we have mentioned (p. 453), joins the vagus just after emerging from the skull.

How to test the vagus.—Paralysis of the vagus is chiefly evidenced in its palatine and laryngeal branches.

1. The **palate.**—Ask the patient whether he is troubled with the regurgitation of fluids through his nose when he tries to swallow. This is a common occurrence in total paralysis of the soft palate, owing to defective elevation of it during swallowing. For a similar reason the patient is unable to pronounce words which require complete closure of the nasopharynx. Thus "egg" becomes "eng," "rub" becomes "rum," and so on. In unilateral paralysis these symptoms are not observed.

For direct examination of the soft palate place the patient facing the light with his mouth open, and introduce a tongue depressor. The position of the uvula is quite unreliable as a guide to the state of the soft palate. Deviation of it is not uncommon even in health. One must watch the movements of the palate during phonation. Ask the patient, therefore, to say "Ah," and observe whether both sides of the palate arch upwards; in health a sort of depression appears in the centre of the palate when the patient says "Ah." If one side is paralysed that side will remain flat and immobile, and the median raphé will be pulled towards the other side.* In bilateral paralysis the whole palate remains motionless.

2. The **larynx.**—The superior laryngeal branch of the vagus is sensory for the larynx above the level of the true cords, and is motor for the cricothyroid muscle. Unilateral paralysis of the nerve does not produce any symptoms. Bilateral paralysis causes the vocal cords to be relaxed. The voice is therefore hoarse and deep, and the utterance of high notes impossible.

The recurrent laryngeal branch supplies sensation to the larynx below the level of the cords, and motor fibres to all the laryngeal muscles except the cricothyroid. Paralysis of it leads to appearances which are recognised by the laryngoscope, and are described at p. 525.

The **eleventh.**—The accessory part of this nerve gives to the vagus its motor fibres for the larynx and pharynx. Lesions of the accessory part, therefore before its junction with the vagus may give rise to laryngeal paralysis. The spinal part of the nerve dips below the sterno-mastoid muscle about one inch below the tip of the mastoid process, and emerges from underneath that muscle again at about the middle of its posterior border. It supplies the sterno-mastoid and trapezius, which are also supplied by twigs from the cervical plexus. Lesions of the eleventh nerve, therefore, lead to paralysis of these muscles.

How to test the spinal accessory— Paralysis of the upper part of the trapezius is evinced by asking the patient to shrug his shoulders while the observer offers passive resistance by pressing on the shoulders from behind. Paralysis of the sternomastoid causes difficulty in rotation of the chin towards the opposite side.

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* The manner in which the palate rises in such a case has been compared to the ascent of a curtain of which one string is broken.

The **twelfth or hypoglossal nerve.**

Anatomy.—The twelfth nerve arises from a nucleus in the lower part of the floor of the fourth ventricle, close to the middle line. It emerges between the anterior pyramid and the olive. It is a purely motor nerve, supplying the tongue and the depressors of the hyoid bone. Its cortical centre is in the lower part of the ascending frontal convolution.

Test.—Ask the patient to put out his tongue as far as possible. If the hypoglossal is paralysed the tongue, instead of being protruded straight, is pushed over to the paralysed side. Be careful not to mistake an apparent deviation of the tongue, really due to the mouth being twisted to one side, for a real deviation of it. Such an apparent deviation occurs in facial paralysis. Ask him also to move his tongue from side to side, and to lick each cheek with it; observe whether he can do so freely. Note whether there is any wasting of the tongue, and whether there be any tremor or fibrillary twitching in it. The presence of wasting indicates that the lesion is either nuclear or infranuclear.

Paralysis of the cervical sympathetic may be conveniently considered here. A complete description of the functions and distribution of the nerve, however, is not necessary in such a work as this. For purposes of diagnosis the fibres supplied to the eyeball are alone of importance. These probably take origin in the anterior part of the floor of the aqueduct of Sylvius, pass down the cervical cord to a centre in the lower cervical and upper dorsal regions (ciliospinal centre), from which the fibres emerge in the last cervical and first dorsal nerve roots and pass to the sympathetic cord by the rami communicantes. From the cervical sympathetic cord the fibres pass along the internal carotid to the cavernous plexus, and thence *viâ* the ophthalmic division of the fifth to the eyeball. They convey the impulses which cause dilatation of the pupil, and supply also the unstriped muscle in the insertion of the levator palpebræ into the upper lid. Paralysis of the cervical sympathetic is recognised by the following signs :—Some recession of the eyeball, so that the eye looks smaller than its fellow ; slight drooping of the upper lid, due to paralysis of the unstriped muscle fibres contained in it ; contraction of the pupil with absence of dilatation on shading the eye, or on instillation of cocaine ; abolition of the ciliospinal reflex ; absence of sweating, even after the use of pilocarpin, on the corresponding half of the head and neck both in front and behind extending as low as the third rib and third dorsal spine, and over the whole of the upper limb on the same side.

SECTION IV.—MOTOR FUNCTIONS.

In investigating the motor functions of a patient, one has to satisfy himself on four separate points—

I. Is there any muscular paralysis or weakness?

II. Can the patient co-ordinate his actions normally?

III. What is the state of nutrition of his muscles?

IV. Is there any abnormal muscular movement present?

I. **Investigation of motor power.** — The first thing to be noted as regards the patient's voluntary power is whether or not he is capable of performing gross muscular movements. Can he walk? Can he sit up in bed? Can he move each of his limbs as a whole?

These main points having been determined, it may be necessary to investigate the principal muscles and groups of muscles separately.

The general rule for one's guidance in this investigation is to ask the patient to throw into action the particular muscle or group of muscles which one wishes to test, whilst the observer offers to that action a greater or less degree of passive resistance. The following is the method of procedure for the chief muscles which one wishes to test :—

1. **Upper limb.**

Flexors of fingers.—Ask the patient to squeeze your hand. If a record of the power of grasp be desired, which can be compared with the result yielded in similar circumstances on another occasion, one should make use of the dynamometer.

Interossei and lumbricales.—Paralysis of these muscles gives rise in cases of some standing to a peculiar position of the hand known as "**main en griffe**" or claw-hand. The above-mentioned muscles produce flexion of the first phalanges on the metacarpals and extension of the other two phalanges. Paralysis of them produces, by over-action of their opponents, over-extension of the first phalanges and flexion of the other two. The fingers are also slightly separated from one another. Claw-hand occurs in some cases of progressive muscular atrophy; and, in a partial form, in paralysis of the ulnar nerve. Claw-foot is an analogous condition.

Opponens pollicis.—Ask the patient to touch the tip of his little finger with the point of his thumb.

Adductor of thumb.—Ask the patient to grasp a book between the forefinger and thumb, keeping the thumb and fingers in the same plane.

Flexors of wrist.—The hand being held with the palm upwards, ask him to bring the points of his fingers towards the front of the forearm.

Extensors of wrist.—The hand being held with the palm downwards, the observer grasps the patient's wrist and asks him to bend the hand up backwards as far as possible. If he is unable to produce dorsiflexion of the wrist some weakness or paralysis of the extensors is present.

Slight weakness of the extensors of the wrist may be elicited by asking the patient to grasp something firmly in his hand. If the extensors are weak the wrist becomes flexed as he does so, owing to the flexor muscles getting the better of the extensors.

Weakness or paralysis of the extensors of the wrist leads to the condition known as **wrist-drop.**

Supinator longus.—Place the arm midway between the prone and supine positions; then ask the patient to bend up the forearm whilst the observer offers opposition to the act by grasping the hand. If the muscle be healthy, it will be seen and felt to stand out prominently at its upper part.

Biceps.—The patient's elbow being held against his side, ask him to bend up the forearm while opposition is offered by grasping the hand or wrist. If the biceps is healthy it will be observed to stand out prominently as it contracts.

The *Triceps* is tested by asking the patient to straighten out his forearm whilst the observer endeavours to keep it flexed by means of passive resistance.

Deltoid.—Ask the patient to lift his arms straight out at right angles to the trunk. In paralysis of the deltoid he is unable to do so.

Pectorals.—Ask the patient to stretch his arms out in front of him, and then to clap his hands while the observer endeavours to hold them apart. Note whether both heads of the muscle are thrown into contraction or not.

Serratus magnus.—Ask the patient to push against resistance. In a healthy condition of the muscle its various digitations will be seen to stand out in contraction, whilst the scapula will remain in close apposition to the chest wall. If the muscle is paralysed, the posterior border and inferior angle of the scapula will come to project more or less when the patient pushes.

Latissimus dorsi.—Ask the patient to clasp his hands behind his back while the observer, standing behind the patient, offers passive resistance to the downward and backward movement, or grasp the two posterior axillary folds and ask the patient to cough. In health the latissimus can be felt to contract.

2. **Trunk muscles.**

Weakness of the muscles of the abdomen is indicated by the patient being unable to raise himself in bed without the aid of his arms.* To test the *erector spinæ* and muscles of the back, make the patient lie on his face and try to raise his head from the bed by extending the neck and back. If the back muscles are healthy, they will be seen to stand out prominently during this effort.

The method of detecting paralysis of the *diaphragm* has already been described (p. 253).

The *trapezius* is tested in its upper part by asking the patient to shrug his shoulders while the observer tries to press them down from behind. In its lower part it can be tested by asking him to approximate the shoulder blades.

3. The **head muscles.**—For the methods of detecting weakness or paralysis in the muscles of the head, the reader is referred to the section dealing with the investigation of the Cranial Nerves (p. 422).

4. The **lower limb.**

The muscles of the foot are tested on the same lines as the corresponding muscles of the hand—passive resistance being offered to their action in each case.

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* Babinski's "rising-up sign" consists in making the patient lie on his back with the legs extended and rise up without using his hands. In *organic* paralysis of a leg the affected limb will rise first, owing to slight rigidity; but in *functional* paralysis this does not occur.

Extensors of knee.—Bend up the patient's knee, and then, pressing with your hand on the sole of his foot, ask him to try to straighten it out again.

Flexors of knee.—Turn the patient on his face, and then ask him to bend up the knee whilst the observer endeavours to hold it down by pressing upon the back of the ankle.

Extensors of thigh.—The knee being extended, lift the patient's foot off the bed, and ask him to depress it against resistance. If the extensors of the hip are paralysed he will be unable to do so.

Flexors of thigh.—The knee being extended, ask the patient to raise his leg off the bed.

The *adductors of the thigh* are tested by abducting the limb, and then asking the patient to bring it back to the middle line while passive opposition is offered to the act. In a similar way the *abductors* are tested by bringing the limb across the middle line, and then asking the patient to move it outwards again.

Rotators of the thigh.—Turn the patient on his face, and bend the knee to a right angle. Then ask him to roll the leg outwards or inwards whilst passive resistance is offered by grasping the foot.

If, on carrying out any of these tests, a muscle or group of muscles is found to have only a feeble power of contraction, **paresis** of it is said to be present. If no contraction be elicited at all the condition is one of **paralysis.**

The term **hemiplegia** is applied to a condition in which there is paralysis of one side of the face and of the arm and leg on the same side If the paralysis of the arm and leg be on one side, and that of the face on the other, the condition is one of **crossed paralysis.** The term **paraplegia** is applied to a paralysis of the lower part of the body; the term **monoplegia** to a paralysis of one arm (which is therefore characterised as a brachial monoplegia) one leg (*crural* monoplegia), or one side of the face (*facial* monoplegia).

The detection of paralysis—and still more of paresis—in a patient who is comatose is often a very difficult matter. It is to be observed, however, that even in such a patient one can usually detect a greater degree of *limpness* in the paralysed limb. If the arm, for example, be raised from the patient's side and allowed to drop, it falls, if it be paralysed, just as if it did not belong to the patient; the sound arm also falls, but not in such an utterly limp fashion. The distinction, however, is often by no means easy.

II. Investigation of muscular co-ordination.

By muscular co-ordination is meant the co-operation of separate muscles, or groups of muscles, in order to accomplish a definite act. If such co-operation be absent or imperfect, the performance of certain acts becomes difficult or impossible, and the condition is then said to be one of **incoordination.** The term **ataxia** or **ataxy** has a similar meaning.

The co-ordination or harmonious action of groups of muscles is the product of various factors, amongst the chief of which are sight, the muscular sense, the contractility and elasticity of the muscles, and in the case of some acts, at least, cutaneous sensibility. If incoordination be present, it is not easy to say which of these factors is at fault. Nor is this necessary, the fact of incoordination being all that one wishes to ascertain. It is usual, however, to eliminate the sense of sight, as the help it affords the patient is so great that it might prevent slight degrees of incoordination from being detected. The eyes should therefore be bandaged.

How to test co-ordination.

1. *In the upper limbs.*

The eyes being bandaged, ask the patient to touch the point of his nose first with one forefinger and then with the other. Or ask him to bring the points of the two forefingers together. If he is able to do these things successfully, without making random shots, no incoordination is present.

Another good test of co-ordination in the upper limb is to ask the patient to thread a needle. In this case, of course, the eyes must be left uncovered.

2. *In the lower limbs.*

If the patient is able to walk, a good test for co-ordination in the lower limbs consists in asking him to walk along a straight line—*e.g.* a crack between two boards of the floor, or the edge of a carpet. If incoordination is present he will soon deviate to one side or the other.

If he cannot walk, proceed as follows: The eyes being covered, ask the patient, as he lies in bed, to touch the dorsum of one foot with the great toe of the other.

Another method is to leave the eyes open and then to ask him to follow with his toe one's forefinger, with which one describes circles in the air. If he is able to describe the circles accurately his power of co-ordination is good.

A special test for co-ordination in the lower limbs is to make the patient stand with his heels together and his eyes shut, and to notice whether he stands steadily or sways about.* The inability to do this, which exists in locomotor ataxy, is known as **Romberg's symptom.** It is due in part usually to the absence of sensation in the soles of the feet, but is found even when sensation is unimpaired.

III. The **state of nutrition of the muscles.**

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* To elicit slighter degrees of the phenomenon it may be necessary to make the patient stand on tip-toe with the knees bent.

—This is gauged roughly by pinching the muscles and noting whether they are firm, as in health, or wasted and flabby. In the disease known as pseudo-hypertrophic paralysis some of the muscles are abnormally firm and large. This is especially apt to be the case with the calf muscles and the infraspinati. Such hypertrophy must not be mistaken for a mere increased muscular development. It is due to an overgrowth of the interstitial tissue of the muscle at the expense of the muscle fibres, and is really a sign of disease. The finer indications regarding muscular nutrition which are afforded by the use of electrical stimulation are described in the section on the Electrical Examination of Muscles and Nerves (p. 486).

IV. **Abnormal muscular movements.**—These consist of involuntary muscular contractions of various sorts. The first thing to note is whether the movements are widespread or localised.

If they are confined to one part of the body, note the joints at which the movements occur, and the muscles or groups of muscles involved. The term **spasm** is often applied to any exaggerated and involuntary muscular contraction. The contraction may either be continuous, in which case it is said to be "**tonic,**" or there may be a series of short contractions with complete or partial relaxation of the muscle in the intervals; in that case they are spoken of as "**clonic.**"

A general increase of "tone" in the muscles is spoken of as **hypertonia**; a general diminution as **hypotonia.** **Kernig's sign** is a phenomenon depending upon the existence of an increased tonus. In order to elicit it as originally described by its discoverer* place the patient on his back with the legs relaxed and extended at the knees. On raising him to a sitting posture the knees become flexed, and cannot be straightened when he is again laid upon his back, owing to a strong contraction of the hamstrings. The sign is more commonly elicited now by flexing the thigh to almost a right angle, and then trying to extend the knee (Fig. 129), whilst the other leg is kept flat upon the bed. If a positive result is obtained the hamstrings are thrown into contraction.

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* *Berlin. Klin. Woch.*, No. 52, 1884.

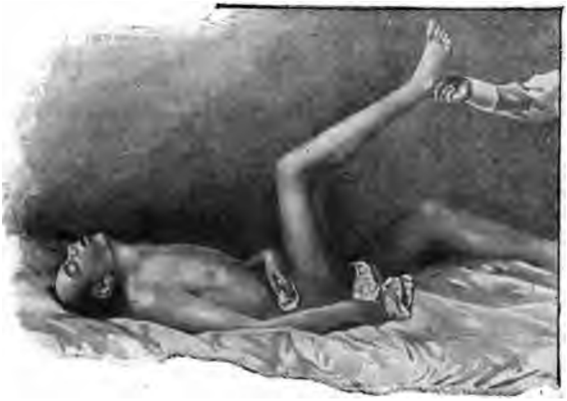

Fig. 129.—Head retraction and Kernig's sign.
(After a photograph by Dr. W. Thyne.)

Kernig's sign is present in about 85 per cent. of all cases of meningitis, but has also been found occasionally in cerebellar hæmorrhage and other conditions at the base of the brain. It commonly coexists with cervical opisthotonos.

The term **contracture** or **rigidity** is usually applied to that continuous spasm of groups of muscles often observed in cases of paralysis due to cerebral lesions. It is doubtful, however, whether this is a true muscular contraction.

Tetanic spasm is observed in its completest form in tetanus, strychnine poisoning, hydrophobia, and some forms of hysterical fits. It may lead to a bending of the whole body backwards (*opisthotonos*) or sideways (*pleurosthotonos*) or forwards (*emprosthotonos*).

The term **tetany** is applied to a peculiar form of tonic spasm affecting the hands and feet. The fingers are pressed together, flexed at the metacarpo-phalangeal joints, but extended at their phalangeal articulations. The thumb is tucked in under cover of the fingers. The hand therefore assumes a conical shape, and is sometimes said to be in the "obstetric position," that being the position in which the accoucheur holds his hand when introducing it into the vagina. The wrist is also flexed. In the foot one finds marked flexion of the toes and anterior part of the foot, sometimes also of the ankle. Tetany is chiefly a disease of infancy, but is sometimes met with in adults who have dilatation of the stomach.

Clonic spasms are of various degrees of severity. If very widespread they are spoken of as **convulsions,** and are seen typically in epilepsy. If the patient gives a history of fits, their character should be inquired into, following the lines laid down on p. 10. Should the observer be fortunate enough to witness an attack he should note:—

1. *The nature and distribution of the movements.*—Are they general or confined to one limb or part of a limb? What part is first and what last affected? Are the convulsions tonic or clonic? Is there any struggling, arching of the back, or attitudinising? Are the abdominal muscles involved or not?

2. Is there any *involuntary evacuation* of the bladder or rectum? Is there any blood or froth about the mouth?

3. *The state of the eyes.*—Is the conjunctival reflex present or abolished? Do the pupils re-act to light? Is there any inco-ordinate movement of the eyeballs?

4. How does the patient behave *after the fit?*

If one group of muscles is first affected, the spasm spreading to others by degrees, it indicates a spread of the irritation along the surface of the cortex cerebri, and occurs typically in Jacksonian epilepsy. The mildest variety of clonic muscular contraction is termed **tremor.**

Tremor may be either *fine* or *coarse.* Fine tremor is usually more easily felt than seen. It occurs in exophthalmic goitre, alcoholism, and in some forms of metallic poisoning. All forms of tremor are most easily seen by increasing the leverage at which the affected muscles act. Thus tremor of the upper limbs is often brought out by getting the patient to extend his arms in front of him. In describing tremor, always note whether it is constantly present or whether it is affected in any way by voluntary muscular action. Ask the patient to lift a glass of water to his lips, or to attempt to follow the movements of your finger, and note whether the tremor is increased thereby (as it is, for example, in cases of disseminated sclerosis), or whether it is diminished or altogether abolished.

Tremor which only comes on when the patient attempts to use the affected muscles is sometimes described as "*intention*" *tremor.*

Clonic contraction of individual fibres or bundles of fibres in a muscle is termed **fibrillary twitching.** It is seen in many cases of progressive muscular atrophy, and indicates an abnormal state of nutrition in the spinal cells connected with the affected fibres.

The term **choreic** is applied to involuntary movements of a purpose-like character occurring in individual muscles or groups of muscles. Such movements are seen most typically in chorea minor or St. Vitus's dance. They consist of abrupt involuntary twitchings or contractions which cause the patient (usually a child) to seem fidgety and unsettled. They are increased by mental agitation, but are often diminished by voluntary muscular effort. If the movements are limited to one side of the body the term *hemichorea* is applied.

Choreic movements, if slight, can be elicited in two ways. Firstly, one may ask the patient to hold both hands straight up above the head; or, secondly, one may ask him to spread out his hands, palms downwards, on the extended hands of the observer. In the former case it may be observed that the patient is unable to hold up his hands steadily for any length of time; in the latter one may notice that little twitchy movements soon become evident in the patient's fingers.

If the patient is able to write at all, one may get him to scrawl his name with the affected hand, and keep the result for purposes of comparison later. In this way one is able to gauge any increase or diminution in the choreic movements.

The last point to be noted regarding any abnormal muscular movement is whether or not it persists during sleep.

The term **athetosis** is used to describe a slow muscular contraction which leads to continuous and deliberate twisting movements, specially affecting the hands and feet.

SECTION V.—SENSORY FUNCTIONS.

In investigating the sensory functions of a patient, we have to test the acuteness of the following forms of sensibility :—

1. Common sensibility. This includes the powers *of appreciating* touch and pressure.

2 Sensibility to pain.
3. The temperature sense.
4. The muscular sense.

In addition, one has to note the presence or absence of any abnormal sensations.

At the outset it is well to explain to the patient the nature of the tests to be performed, so as to secure, as far as possible, his intelligent co-operation. The eyes should then be bandaged, and the different forms of sensibility tested as follows:—

1. **Common sensibility.**

(*a*) **Touch.**—The point of the finger or, preferably, the feather end of a quill pen may be used as a stimulus. For carefully mapping out areas of altered sensibility a small cone of cotton wool is best. It is so light that the element of pressure is entirely eliminated. A fine camel-hair brush also answers the purpose very well.

Tell the patient to say "Now" whenever he feels a touch. Compare corresponding points on opposite sides of the body, and employ every now and then a negative test, asking the patient if he feels you touch him, in order to prevent his making random replies. Ask him also to localise the stimulus by pointing to the exact position of the spot touched. This is important, as a patient may be able to feel the stimulus and yet not be able to localise it.

Sensibility to touch may be altered in various ways. (1) It may be entirely abolished. This constitutes **anæsthesia.** If the abolition affects the whole of one side of the body, it is termed *hemianæsthesia.* If the existence of anæsthesia be discovered, one must at once proceed to mark out its exact extent and boundaries. (2) It may be exaggerated, so that what should in health be felt as a mere touch produces a painful impression resembling pricking or burning. *This constitutes* **hyperæsthesia.** If hyperæsthesia be discovered, its extent should be carefully mapped out. Hyperæsthetic spots are sometimes met with, especially in hysterical patients. The commonest sites for these are over the brim of the pelvis, in the inframammary region, along the vertebral column, and on the scalp. Pressure on such spots may sometimes induce hysterical fits. If that occurs, the spots are spoken of as "hysterogenetic." (3) Sensation may be appreciated well enough, but there may be great delay in its conduction, an appreciable interval occurring between the application of the stimulus and the response of the patient. This **delayed conduction** exists not unfrequently in cases of alcoholic neuritis and locomotor ataxy. (4) The stimulus may be badly localised, the patient believing, for example, that the outer side of a limb was touched when the stimulus was really applied to its inner aspect. Sometimes a touch on one side of the body is referred to a corresponding point on the opposite side. This is termed **allocheiria.**

(*b*) **Pressure.**—This is tested by supporting the limb or part to be tested in such a way as to eliminate any muscular action on the part of the patient, and then laying different weights on the surface, and asking the patient which feels heavier. Different coins of the same size—*e.g.* sovereign and farthing—answer the purpose well enough.

2. **Sensibility to pain.**

The point of a quill pen may be used as a stimulus. The point of a pin or needle has the disadvantage of being so fine that it may miss the nerve endings. The application of a faradic current is also an excellent method of testing sensibility to pain. It enables one to gauge degrees of sensibility by noting what strength of current is required to cause pain, and then comparing the result with the corresponding area on the opposite side.

Absence of the sense of pain is termed **analgesia;** an exaggerated sensibility to pain, so that a mild stimulus produces an unusual degree of suffering, is termed **hyperalgesia.**

3. **Temperature sense.**—Use test tubes of hot and cold water. Touch the part to be investigated with each in turn, and ask the patient whether it feels hot or cold. Note whether he calls hot "cold" or cold "hot," or whether he does both.

It should be noted that the different forms of sensibility already mentioned may require to be tested on the accessible mucous membranes as well as on the skin surfaces. The sensibility of some viscera is also of importance. Thus the absence of pain on squeezing the testicle may be an early sign of locomotor ataxy.

4. **Muscular sense.**—This demands the investigation of two separate points.

(*a*) The patient's power of estimating weights.

(*b*) His power of appreciating the position of his limbs.

(*a*) **Sense of weight.**—Place in the patient's hand substances which resemble one another as far as possible in every respect except as regards weight. Metal balls covered with leather, some being solid and others hollow, are often used for the purpose. In their absence one may use two match boxes, one full, the other empty, or some other extemporised device. A solid ball and a hollow one may be placed one in the patient's right hand and the other in his left, and he is then asked to state which is the heavier; or one hand may be tested at a time, the balls being lifted one immediately after the other. If the leg is being investigated, the weights should be placed in a handkerchief and slung round the patient's ankle.

(*b*) **Sense of position.**—The patient's eyes *being carefully* shut, take hold of one of his limbs and move it about in various directions through the air, finally leaving it in some definite position, say semiflexed and slightly elevated; then ask him to put the corresponding limb in a similar position. If there be no paralysis of the latter and yet the patient is unable to imitate with it the position of the other, then there is reason to believe that the sense of position is impaired.

In testing a patient's sense of position in this manner, be careful not to allow the part tested to touch any other skin surface; otherwise, the patient will be able to appreciate its position by the information derived from his ordinary sense of touch.*

Are there any abnormal sensations present?—These are termed **paræsthesiæ**, and consist in various sensations experienced by the patient in the absence of any outward stimulus. The commonest of these are a feeling of "needles and pins," of numbness, of heats or chills, of pressure or tightness (a good example of the latter being the "girdle pain" of locomotor ataxy), of itching—sometimes termed "pruritus"—or a feeling as if insects were crawling over the body (formication).

The term **aura** is applied to the curious paræsthesiæ which frequently precede an epileptic fit and serve as a warning of its approach.

SECTION VI.—REFLEXES.

There are three classes of reflexes which one has to test.

1. The superficial reflexes;

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* A very delicate test for defective muscular sense (and therefore for inco-ordination) in the upper limbs consists in shutting the patient's eyes and then making him hold his arms straight out in front of him with the fingers in a horizontal row. After a moment or two, if the muscular sense be defective, the fingers cease to remain in an even line. Some will rise a little, others fall, or even become twisted in below the rest.

2. The deep or tendon reflexes;
3. The organic reflexes (including the action of the sphincters).

We shall consider these separately.

1. SUPERFICIAL REFLEXES.

In these the simplest form of reflex action is concerned. On stimulation of a certain part of skin or mucous membrane contraction of certain muscles results. The path of the impulse is by the sensory nerve fibres to the grey matter of the cord or a sensory centre in the brain, thence by motor nerve fibres to the muscle. A lesion in any part of this path causes the reflex to disappear. Thus anæsthesia of the skin, disease of the sensory fibres or posterior nerve roots, changes in the grey matter of the cord, lesions of the motor nerve fibres or of the fibres of the muscles, may all cause abolition of the superficial reflexes. In addition to this, it must be borne in mind that the reflex excitability of some individuals is normally very much greater than that of others, and this makes it difficult for one to estimate the value of slight alterations in the reflexes unless the lesion is unilateral, in which case the healthy side can be taken as a standard of comparison. The investigation of the superficial reflexes is of more value as affording information regarding the health or otherwise of the reflex arc concerned than as a guide to the presence or absence of disease elsewhere. In hemiplegia the superficial reflexes are usually diminished on the healthy side. Exaggeration of both knee jerks along with diminution of the plantar reflexes is a combination characteristic of many cases of functional paraplegia.

The chief superficial reflexes of spinal origin, their nature, *the mode* of obtaining them, and the level of the cord concerned in their production, are given in the table on p. 477.

The **Plantar reflex** demands special consideration. In order to elicit it the patient should be in the supine position, with the lower limb semi-flexed at the hip and knee and rotated outwards, the knee resting on a pillow. This induces muscular relaxation, which may be increased by distracting the patient's attention by conversation or by making him perform with his hands some act which requires attention. Care should be taken that the sole of the foot is warm and dry. The plantar region is stimulated by gentle scratching with the finger-nail or with a soft quill. In healthy adults a minimal stimulus produces a contraction of the tensor vaginæ femoris, often accompanied by a slighter contraction of the adductors of the thigh and sartorius.* With a slightly stronger stimulus flexion of the four outer toes appears, which increases with the strength of the stimulus till all the toes are flexed on the metatarsus and drawn together, the ankle being dorsiflexed and inverted. With still stronger stimuli violent irregular movements of the limb occur, which spread to the lower part of the trunk and to the opposite side. The position of the foot at the height of a response to a moderate stimulus is shown in Fig. 130 ("flexor response").

It is doubtful whether the plantar reflex is ever constantly and completely absent in healthy subjects.

In infants up to the age of learning to walk the reflex is very brisk, and differs markedly from that in adults. The earliest response is in the great toe, which is drawn back. This is followed by extension and spreading out of all the toes, with eversion of the foot or dorsiflexion of the ankle, and subsequently flexion of the hip and knee ("infantile response").

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* *See* an admirable paper by Dr. James Collier in *Brain*, 1899, part 85, from which most of the above account has been *compiled.*

During sleep the plantar reflexes are diminished, and the infantile and adult forms preserved, except in some children up to the age of twelve years, where in deep sleep the infantile form of reflex returns. In pathological conditions the reflex varies, and may be of great diagnostic importance. In *lesions of the pyramidal systems* an alteration in the type of response was first described by Babinski.† In this, which is spoken of as **Babinski's sign**, or the "extensor response," the reflex closely resembles that obtained in infants, but differs in a few points. The whole response is more deliberate than that obtained either in adults or infants, and appears with much

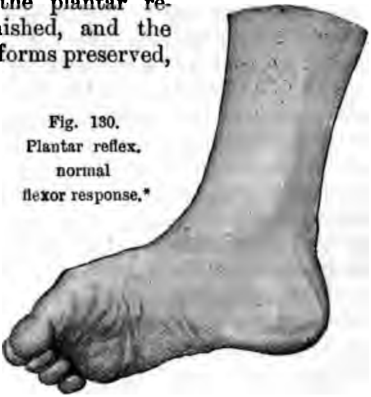

Fig. 130.
Plantar reflex, normal flexor response.*

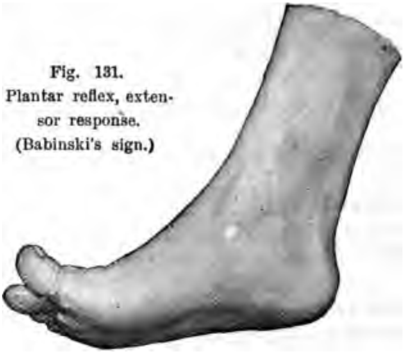

Fig. 131.
Plantar reflex, extensor response.
(Babinski's sign.)

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* After instantaneous photographs by Dr. James Collier.

† *La Semaine Médicale*, Juillet 27, 1898.

more certainty than does the flexor response to each stimulation. Extension of the great toe precedes all other movement. It is followed by extension of the other toes and by dorsiflexion of the ankle. The tensor vaginæ femoris does not contract early. The small amount of movement at the ankle is conspicuous, and contrasts with the brisk movement at the ankle in the ordinary response. The extensor response is often more easily elicited by stimulation of the outer part of the sole, whereas the flexor response is usually more easily obtained by stimulating the inner part. Fig. 131 shows the position of the foot at the height of a moderate extensor response.

The extensor response is only met with in adults in cases of organic disease involving the pyramidal systems, and in cases of total transverse lesion of the cord is the only reflex phenomenon present in the lower limbs. In functional cases, if the plantar reflex can be elicited at all, the flexor response is obtained. In tabes and peripheral neuritis the same is true. In neurasthenia, chorea, myopathies, poliomyelitis, and paralysis agitans the flexor response is found; also in intracranial tumours, provided the pyramidal systems are not involved.

The following superficial reflexes are dependent on cranial nerves:—

(1) **Conjunctival.**—Elicited by touching the conjunctiva, resulting in contraction of the orbicularis palpebrarum. The nerves concerned are the fifth (sensory) and the seventh (motor).

(2) **Pupil reflexes.**—(*See* pp. 441 and 502).

(3) **Palate reflex.**—Elevation of the palate on touching the mucous membrane covering it. The nerves concerned are the glossopharyngeal and the *vagus* (or spinal accessory?).

REFLEX.	HOW EXCITED.	RESULT.	LEVEL OF CORD CONCERNED.
Plantar	Stroking sole of foot	Movements of toes, of toes and foot, or leg	Lower part of lumbar enlargement (fifth lumbar and first sacral segments)
Gluteal	Stroking skin of buttock vertically	Contraction of gluteal muscles	Fourth and fifth lumbar segments
Cremasteric	Stroking skin at upper and inner part of thigh*	Drawing upwards of testicle	First and second lumbar segments
Abdominal	Stroking abdominal wall from costal margin to nipple line	Contraction of abdominal muscles	Eighth to twelfth dorsal segments
Epigastric	Stroking side of chest downwards from nipple	Drawing in of epigastrium on same side	Fourth to seventh dorsal segments
Scapular	Stroking skin in interscapular region	Contraction of scapular muscles	Fifth cervical to first dorsal segments

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* The Cremasteric Reflex can often be most easily elicited by pressing over the Sartorius in the lower third of Hunter's canal.

2. DEEP OR TENDON REFLEXES.

If a muscle be put upon the stretch and its tendon be then sharply struck, the muscle immediately contracts. This is spoken of as a *deep* or tendon reflex. It is very doubtful, however, whether one is correct in speaking of it as a reflex at all. It would seem that the contraction of the muscle follows too rapidly on the blow upon the tendon to permit of there being time for a reflex arc being traversed. What really happens is probably this. The stretching of the muscle reflexly increases its "tone." When the tone is thus raised any mechanical stimulus, in this case the blow upon the tendon, by suddenly increasing the tension of the muscle, excites in it a direct contraction. The only point where a reflex act really comes in, therefore, is in the preliminary raising of the muscle tone. Without this increase of tone, however, the subsequent direct contraction would be impossible.

We have already indicated (p. 401) that the "tone" of a muscle is dependent upon the cells in the anterior cornua of the spinal cord—*i.e.* the lower neurons. When the control of the upper neurons over the lower is cut off, the latter produce an increased "tone" in the muscles, with the result that the tendon reflexes are more easily excited than normally. In other words, their reflexes are increased or exaggerated. Exaggeration of tendon reflexes, therefore, is characteristic of lesions affecting the upper neurons—*i.e.* the cerebral cortex, or the fibres passing from it to the anterior horns of the cord. A similar exaggeration may be brought about by anything which stimulates the lower neurons, thus making them more able to resist the controlling *influence of* the upper. Strychnine and the toxin of tetanus are able to do this, and therefore produce an increase of the tendon reflexes.

On the other hand, anything which impairs the activity of the lower neuron will cause a diminution in the tone of the muscles, and will thus make the tendon reflexes correspondingly difficult to elicit. Diminution or abolition of the tendon reflexes is therefore characteristic of lesions affecting the lower or spinal neuron. Of course, any disease of the muscle fibres themselves, or of the sensory fibres which proceed from the muscle to the spinal cord, and along which the stimulus travels when the muscle is first stretched, which stimulus causes the reflex increase of tone, will also cause a diminution of the tendon reflexes by preventing the necessary increase of tone from taking place. Hence it is that in locomotor ataxy, in which the posterior roots are much involved, the deep reflexes are absent.

There is only one point more to be referred to, and it is rather a confusing one. In a lesion—*e.g.* a fracture dislocation—which produced complete transverse destruction of the cord at any level, one might expect that, owing to the cerebral influence being cut off, all the deep reflexes below that level would be exaggerated. This, however, is not the case. The reflexes in that condition are (as a rule) totally abolished. The explanation of this apparent anomaly is still a little doubtful. It has been supposed by some that it is to be attributed to the cutting off of a cerebellar influence which descends to the cells of the anterior cornua, and, so to speak, energises them, so that they are able to maintain the tone of the muscles. It must be admitted, however, that experimental evidence is totally opposed to this conclusion, as lesions of the cerebellum are followed by exaggeration and not by abolition of the deep reflexes.

The **knee jerk** or **pattellar tendon reflex** is the best known of the deep reflexes. It consists in a contraction of the quadriceps extensor when the patellar tendon is tapped. The spinal segments concerned are the third and fourth lumbar.

How to elicit the knee jerk.—If the patient is able to sit up, get him to sit on a chair or on the edge of the bed and cross one knee over the other. If he is unable to effect the latter, pass your wrist under the knee to be tested, resting your hand on the opposite knee and allowing the patient's leg to swing suspended, as it were, on the back of your wrist. If he cannot sit up, bend the knee up a little as he lies on his back and support it by allowing it to rest on your hand or the back of your wrist.

The result of this disposition of the limb is slightly to stretch the quadriceps extensor, and so reflexly to increase its tone. The next thing to do is to try to divert the patient's attention. This may be done by asking him to "let the leg hang as if it did not belong to him," or by engaging him in conversation. The patellar tendon must then be struck a sharp blow midway between the patella and its insertion. The edge of the hand may be used for the purpose, or the edge of a thin book, or the ear piece of a stethoscope, especially if it be surrounded by a rim of solid indiarubber. A percussion hammer is also a good instrument for the purpose.

Immediately after the blow the foot will be observed to be jerked up from the sudden contraction of the muscle.

The briskness of the knee jerk varies greatly in different individuals. In health, however, it is hardly ever entirely absent. Sometimes one is unable to elicit it without having recourse to what is known as "**reinforcement**" of the knee jerk. This consists in asking the patient to make some strong voluntary *muscular effort* with the upper limbs. One may ask him, for example, to hook the fingers of the two hands together and then to pull them against one another as hard as possible. Whilst he is doing so one tries to elicit the knee jerk, and one usually gets it more readily than under ordinary conditions. How reinforcement acts it is a little difficult to say. According to some, it is by increasing the general muscular tone throughout the body; according to others, it acts by diminishing the inhibitory cerebral control.

The following tendon reflexes are similar in nature to the knee jerk, but—probably owing to mechanical difficulties in producing adequate stretching of the muscle—are not usually so easy to obtain in health, and may indeed be entirely absent.

Ankle jerk.—Grasp the dorsum of the foot with one hand and hold up the leg with it. Slightly dorsiflex the foot so as to put the tendo Achillis on the stretch, then with the other hand sharply flick the latter on its under surface. A sharp contraction of the calf muscles results. This reflex can also be conveniently elicited when the patient is kneeling on a chair.

Adductor jerk is produced by abducting the thigh and tapping the tendon of the adductor magnus. Contraction of the adductors results. Sometimes in patients who have very exaggerated reflexes one finds that on tapping the patellar tendon a sudden contraction occurs in the adductor muscles of the opposite thigh. This is termed the "**crossed adductor jerk.**" Its explanation is still rather obscure, but it is apparently a truly reflex phenomenon.

Triceps or elbow jerk.—Flex the elbow to more than a right angle, then tap just above the olecranon. The triceps contracts. The reflex depends upon the eighth cervical and first dorsal segments.

Supinator jerk.—Produced by tapping the supinator tendon just above its insertion into the styloid process of the radius. The fifth cervical is the cord segment concerned.

Wrist jerk is produced by letting the hand hang down, and then striking the extensor tendons just above the wrist. The hand is jerked up. This reflex depends upon the sixth cervical segment.

Jaw jerk.—Ask the patient to open his mouth, but not too widely. Place one finger firmly on his chin and then tap it suddenly with the other hand as in percussion. A contraction of the muscles that close the jaw results. This jerk is never present in health. The motor nucleus of the fifth nerve is the centre involved.

The term "clonus" is applied to the following tendon reflexes:—

Ankle clonus.—To elicit this phenomenon, bend the patient's knee slightly and support it with one hand, grasp the fore part of the foot with the other hand, and suddenly dorsiflex the foot. The sudden strain put upon the calf muscles causes them to contract. The pressure of the hand upon the sole of the foot is meanwhile continued and when the contraction ceases causes the calf muscles again to become tense, and so produces another contraction in the latter. In this way a whole series of contractions—*i.e.* a clonus—results.

The relative tendency to the development of ankle clonus on the two sides is best estimated by slowly dorsiflexing the foot and observing the exact point at which the movements first begin. The less the degree of dorsiflexion required to produce the clonus the greater is the tendency to the development of the latter.*

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* In cases of functional paralysis the clonus may come on *before* the foot has been bent to a right angle; this never happens in cases of organic disease.

Ankle clonus is nearly always a sign of disease. The spinal segments concerned in it are the first to the third sacral.

Knee clonus.—In cases where the knee jerk is exaggerated, one can sometimes elicit a knee clonus by extending the patient's leg and then suddenly pushing down the patella towards the foot. If the pressure on the latter be continued, a series of clonic contractions of the quadriceps can in many cases be produced.

3.—ORGANIC REFLEXES AND SPHINCTERS.

This term includes such processes as respiration, deglutition, micturition, and defæcation. They depend upon complex muscular movements excited either by stimulation of mucous membranes or, in the case of respiration, of a centre in the medulla.

One should always ascertain from the patient whether he has any difficulty in swallowing, noting specially whether there is any regurgitation of food through the nose. The function of **deglutition** does not usually require to be specially tested beyond the examination necessary to exclude the existence of an obstruction (p. 51).

Defæcation.—The patient should be questioned as to any difficulty in the act, and as to the presence or absence of tenesmus. Note also the occurrence or not of incontinence of fæces.

The reflex action of the anal sphincter may be tested by introducing the oiled finger into the anus, and noting whether contraction of the sphincter occurs with normal force, whether it be weak or altogether inactive, or whether there is any spasm excited.

The activity of the anal sphincter reflex may also be tested by pricking the skin in the neighbourhood of the anus. Under normal conditions a brisk contraction of the sphincter should immediately be visible.

Micturition.—The patient should be questioned as to difficulty or pain in the act (*see* p. 9). One should then note whether there is either *retention* of urine or *incontinence* of it. If there be incontinence, ascertain by the use of the catheter whether it be due to the *overflow* from a distended bladder or whether it be a *reflex incontinence*—*i.e.* whether the bladder merely fills up and then empties itself completely by reflex action. In another group of cases the patient feels the desire to micturate, but is unable to restrain the act, which takes place at once. This is spoken of as *precipitate micturition.*

The centres for the bladder and rectum are situated in the third and fourth sacral segments of the cord.

SECTION VII.—TROPHIC FUNCTIONS.

In disease of the nervous system the nutrition of different tissues or organs may be impaired. The *bones* may become more brittle from interstitial absorption, or may exhibit spontaneous fracture (osteopathies), or the *joints* may be the seat of painless effusion with or without atrophy or enlargement of the articular ends of the bones (arthropathies). In other cases the bones and joints are involved together (osteo-arthropathies). More commonly the *skin* is the seat of change. It may exhibit an erythema, which may pass on to ulceration and the formation of bed-sores at points of pressure; pigmentary changes may develop in it, or it may be the seat of various eruptions—urticarial, vesicular, pemphigoid, or herpetic — or it may be simply glossy. Perforating ulcers may appear, usually on the toes or soles of the feet, as in tabes, or there may be actual gangrene, or the development of painless whitlows. In other cases, again, it is the epidermic appendages which especially suffer change, the hair falling out, or the nails becoming dry and brittle. Atrophy of the *muscles* is a common phenomenon.

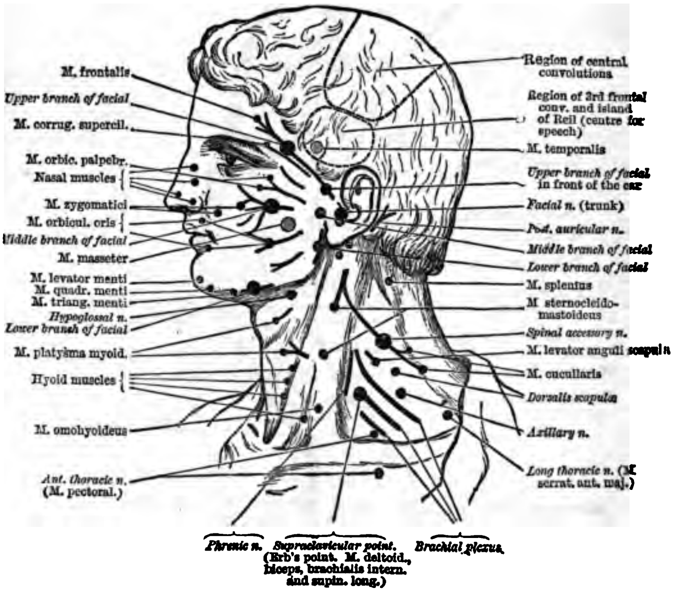

Fig. 132.—Motor points, face and neck.

and may be marked by the appearance of the reaction of degeneration (Section VIII.). More rarely, certain glandular organs, such as the testis, may be the seat of atrophic change.

In taking a nervous case it is necessary to be on the outlook for such changes, and to note their *situation and extent.*

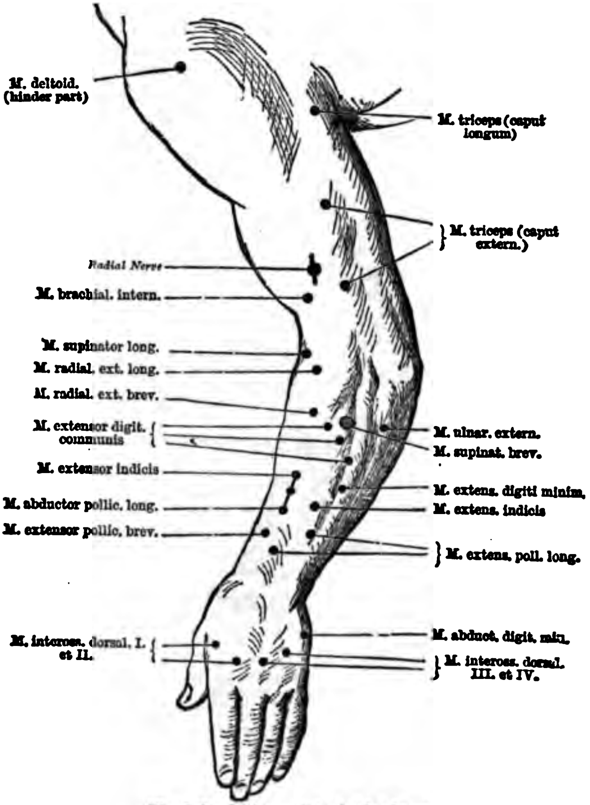

Fig. 133.—Motor points, back of arm.

SECTION VIII.—ELECTRICAL EXAMINATION OF MUSCLES AND NERVES.

1. **Apparatus.**—For the purposes of diagnosis as *distinguished* from therapeutics electricity is applied in

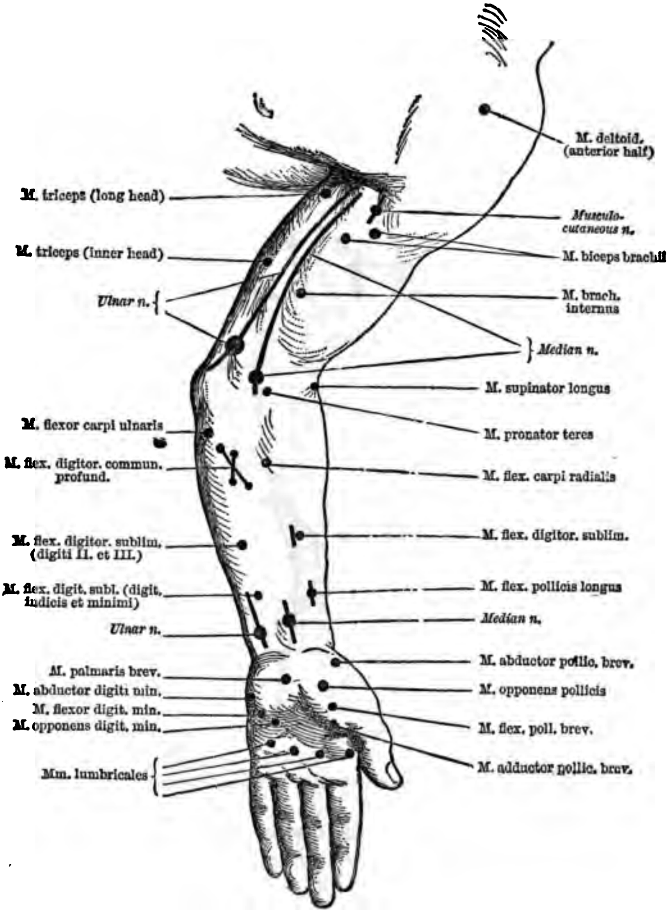

Fig. 134.—Motor points, front of arm.

three forms: First as the continuous current, second as the faradic current, third by using the discharge through Crookes's tubes, with high vacua and suitable internal arrangements, for the purposes of skiagraphy by the X rays described by Prof. Röntgen. The details of this last application are beyond the scope of the present work.

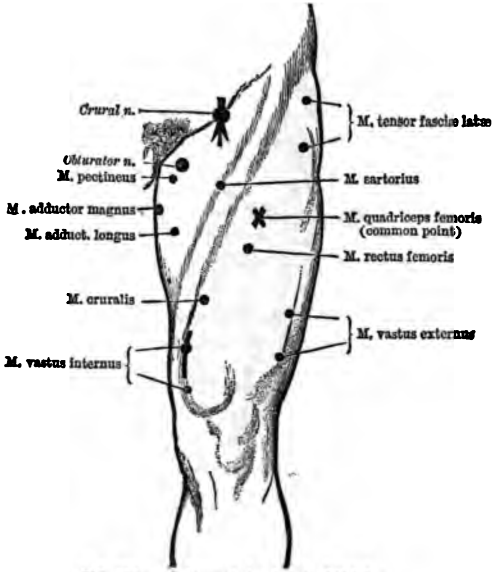

Fig. 135.—Motor points, front of thigh.

The **continuous current** must be of sufficient intensity to overcome the resistance of the body and yet have enough strength left to stimulate the nerves and muscles. It must therefore have an available electromotive force of over 40 volts. A battery of thirty-two Leclanché dry cells is ample for ordinary use, as its electromotive force is decidedly higher than this to start with, and will continue so for a considerable time if properly cared for and neither left too long absolutely idle nor employed to yield very heavy currents which would speedily exhaust the small cells. Where the continuous current from an electric light installation is available it may be used,

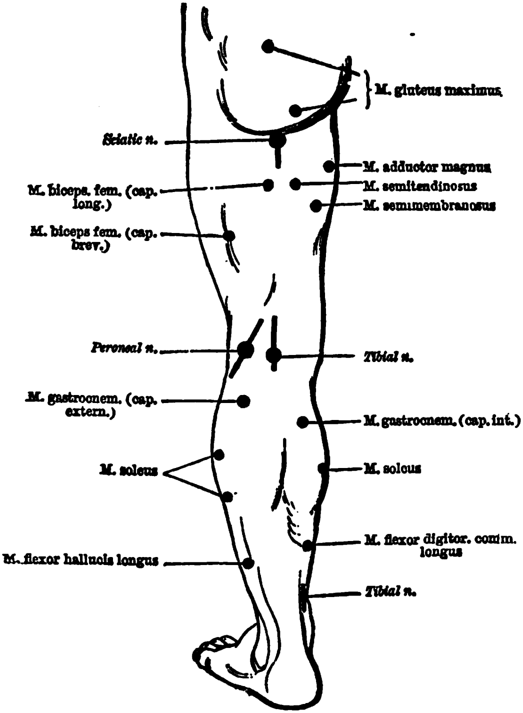
M. gluteus maximus
Sciatic n.
M. adductor magnus
M. biceps. fem. (cap. long.)
M. semitendinosus
M. semimembranosus
M. biceps fem. (cap. brev.)
Peroneal n.
Tibial n.
M. gastrocnem. (cap. extern.)
M. gastrocnem. (cap. int.)
M. soleus
M. soleus
M. flexor digitor. comm. longus
M. flexor hallucis longus
Tibial n.

Fig. 136.—Motor points, back of thigh and leg.

when precautions are taken for the safety of the patient by the employment of shunts to reduce the voltage to a suitable figure.

Whether battery or light installation with regulating board be used, it is of great importance to have efficient accessory apparatus in the way of current reversers, galvanometers, connecting wires, electrodes. The galvanometers should give readings in milliampères, not in unmeaning graduations, as too often *is the case* in those supplied with medical batteries.

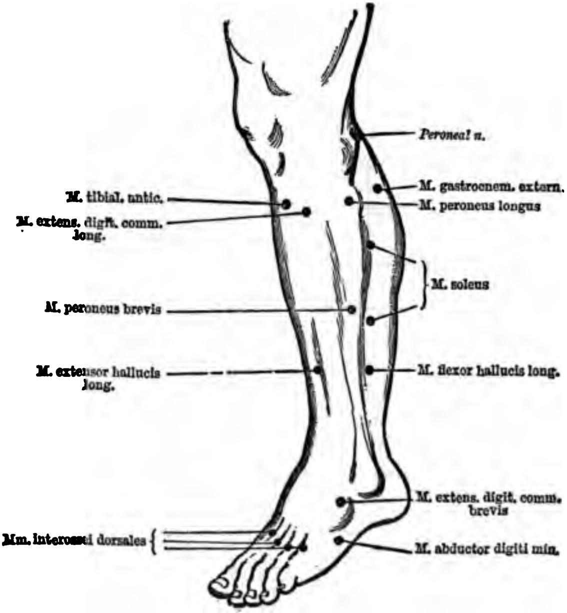

Fig. 137. -Motor points, side of leg.

For the **faradic current** the best form of apparatus is an induction coil of the sledge pattern with a secondary coil of wire which should not be very fine, as very thin wire on the secondary coil produces too great an effect on the sensory, and too little on the motor nerves for most diagnostic purposes. To drive the coil one or two dry Leclanché cells are sufficient. It is often convenient to be able to pass from the continuous to the faradic current, and *vice versâ,* without changing the connections leading to the patient. This can be effected by a suitable switch.

One large flat electrode should be procured, and several small ones, varying in size from a small spherical bulb to a disc of metal with a surface of about 60 or 100 square centimetres. These should be covered with washleather, which can be renewed at frequent intervals, and should be made to screw into a holder with a button by means of which the observer can interrupt the current.

2. **Electro-diagnosis.** (Figs. 132–139.)

Begin with the faradic current.* It stimulates the nerves directly, the muscles only indirectly through their nerve supply. Examine systematically each nerve trunk in the area under consideration, and the motor point of every muscle supplied by each of these nerves. The motor points correspond for the most part with the points of entry of the motor nerves into the muscles which they supply, and are shown in Figs. 132–139. The observer should try the current upon himself before applying it to the patient, both to reassure the latter and to compare the effects it produces on himself with those elicited in the patient. The examination must be conducted in a good light, and the patient must be so placed that access to both sides is easily secured. Before examining any group

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* In dealing with children it is almost always necessary, where a careful electrical examination has to be made, to give an *anæsthetic, although* the anæsthesia need not be very deep.

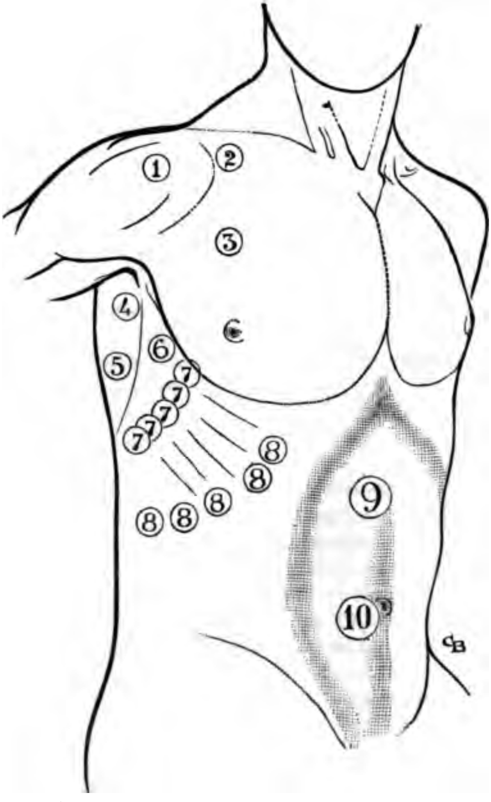

Fig. 188.—Motor points of the trunk anteriorly.

1, deltoid (anterior part); 2, nerve to pectoralis major; 3, pectoralis major; 4. teres major; 5, latissimus dorsi; 6, nerve to serratus magnus; 8, obliquus externus; 9, rectus abdominis (upper part); 10, rectus abdominis (lower part).

of muscles care must be taken that they are so far *relaxed as to* be able to respond readily to stimulation. *A small* electrode—either the button electrode or the disc of 10 to 20 square centimetres, according to circumstances—should be used for the part under examination, whilst the other (or "indifferent") electrode should be a larger plate placed either on the abdomen or between the shoulders. The electrodes and the skin where the tests are applied should be well soaked with a solution of common salt in warm water, but care

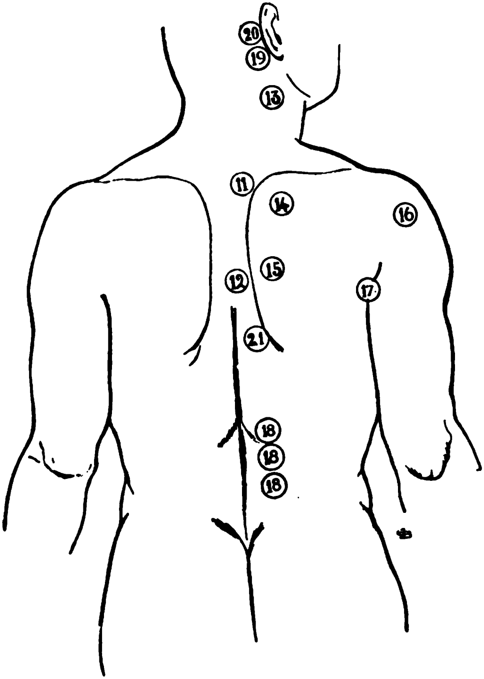

Fig. 189.—Motor points of the trunk, posteriorly.

11 and 12, trapezius; 13, splenius; 14, supraspinatus; 15, infraspinatus; 16, deltoid (posterior part); 17, teres minor; 18, erector spinæ; 19, facial nerve trunk 20, posterior auricular nerve; 21, rhomboid.

must be taken to avoid the presence of any crystals of undissolved salt on the skin.

Note the intensity of the minimum current which produces contraction at each point, and compare the effects of a similar current on the corresponding point on the other side of the body.

The intensity of the current is recorded in terms of the distance of the secondary from the primary coil; battery power, rate of interruption, and other adjustments being assumed to remain constant.

When the faradic current has been employed, pass to **the galvanic.** Use the electrodes in exactly the same manner as for the faradic examination, and, remember, that when a small electrode is used, the nerves and muscles that lie superficially beneath it, and are therefore closer to it, are chiefly stimulated, since response to the stimulus depends on current density. Begin with a weak current, and gradually increase its strength till the muscle responds by slight contraction. At each point employ first the cathode* and then the anode, and observe whether contraction occurs most readily when the circuit is completed or when it is interrupted. Record the strength of the current as indicated by the milliampèremeter and compare it with that which produces similar effects at the corresponding point on the other side.

In health the cathodal closing contraction is the first to appear; in other words, the muscle responds more readily when the pole applied to it is the cathode, and when the electric circuit is completed; a decidedly stronger current is required to elicit the anodal closing and opening contractions, and the cathodal opening contraction appears last of all. This sequence may be represented by the formula

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* *The cathode* or negative electrode is attached to the zinc *rod in the cell,* the anode or positive electrode to the copper or *carbon.*

C.C.C. > A.C.C. > A.O.C. > C.O.C., or by the following expansion of it :—

Weak current	C.C.C.	—	—	—
Medium current	C.C.C.′	A.C.C.	—	—
Fairly strong current	C.C.C.″	A.C.C.′	A.O.C.	—
Very strong current	C.C. Tetanus	A.C.C.″	A.O.C.′	C.O.C.

The contractions in health are abrupt and sharp. Except when the current is very strong, contractions only occur when the circuit is made or broken, not during the passage of a uniform current.

In disease the response to electrical stimulation may be altered either quantitatively or qualitatively. By "**quantitative alterations**" one understands that a given current produces a greater or less contraction than it could were the nerves and muscles in a normal state. "**Qualitative alterations**" involve either or both of two changes—namely, first the character of the contraction, instead of being abrupt, becomes "sluggish"; and second, the cathodal closing contraction is less readily elicited than the anodal contractions.

Such qualitative changes depend on the separation of the nerve and muscle from their nutritive centre. After a brief period the separation induces degeneration, and whilst the degeneration proceeds the nerve first fails to respond to electrical stimuli, and after a longer interval the muscle also becomes insusceptible.

It is during the time that elapses between the insensitiveness of the nerve and of the muscle, that the most characteristic electrical changes are observed, and these are together known as "**the reaction of degeneration.**" They may be summarised as follows :—

I. *Faradic current.*—No response can be elicited, even when a very strong current is employed.

The reason for this is that the faradic stimulus, in consequence of its brief duration, acts only on the nerve, and since the degenerated nerve can no longer transmit stimuli to the muscle, the latter remains unaffected.

II. *Galvanic current.*

1. *Quantitative change.*—The muscular excitability is increased, contraction following the application of a weaker current than is necessary to produce it in health ("The irritability of weakness").

2. *Qualitative changes.*—(*a*) The contraction is no longer sharp, but "sluggish."

(*b*) In most cases anodal closing contraction is elicited with a weaker current than cathodal closing contraction. This phenomenon is less constant and less important than the sluggish character of the contraction.

The quantitative change depends partly on alterations in the nutrition of the muscle, and partly on the removal of inhibitory influences; the qualitative changes are produced partly because the nerve no longer regulates the character of the contraction, and partly also as a result of changes in the muscle itself.

The foregoing description applies to a fully developed reaction of degeneration. This is not manifested till more than a week after the trophic influence has been cut off. The nerves, however, begin to lose their sensitiveness about three days after the injury, and gradually become less and less responsive. The muscles behave as do the nerves to faradic stimulation; to the galvanic current they at first exhibit diminished excitability. After eight or ten days the excitability again increases, and eventually exceeds that found in health. At the same time as the increase appears, the sluggishness of contraction and the abnormal sensitiveness to anodal closure become manifest. When the cause which has led to the severance of the nerve and muscle from their trophic centre cannot be removed, the muscular response once more gradually diminishes, and after the lapse of a considerable period, which may extend to two years or even longer, disappears entirely. When the trophic influence is timeously re-established, the reactions of nerve and muscle progressively return to the normal. In such cases ordinary muscular power usually begins to return some days before the nerves show any response to electrical stimuli.

A new and clinically important form of reaction has been described by Ghilarducci, who has named it the reaction of degeneration at a distance (R.D.d.). In examining for this reaction neither pole is placed on the affected muscle, but the indifferent electrode is situated over the upper dorsal region, whilst the active one is applied at a point further from the trunk than are the muscles to be tested. Thus for the muscles of the forearm it is placed on the wrist, and for the tibialis anticus in front of the ankle. Under these conditions the response occurs almost exclusively at closure, and when the active electrode is negative; whilst the contraction thus elicited is stronger than that which is present in ordinary reaction of degeneration, and can be obtained with a weaker current. Its clinical importance is due to the fact that as it persists after all other electrical stimuli fail to produce any effect, its presence enables one to determine that the muscle is not hopelessly destroyed, but may yet recover within certain limits. If it be absent the observer is justified in concluding that the muscle is wholly severed from its trophic centre.

Ghilarducci regards the phenomenon as due to the increased electrical capacity of the muscle when thus longitudinally stimulated, the increased capacity causing the electric wave to be less abrupt, and thereby prolonging the duration of efficient stimulation.

It will be readily perceived that from the standpoint of diagnosis, electricity yields far more valuable information when the disease is situated in the lower motor neuron, thereby severing the nerve endings and muscles from their trophic centres, than when the morbid process occupies a more central position.

Serious disorder may be present in the higher trophic realms without revealing itself by any change in the electrical reactions—at most there will only be a quantitative change whose detection is often difficult and whose value is uncertain.

In the sensory system electro-diagnosis is of less value, and is chiefly of service in cases where a hysterical element is present.

For recording the results of an electro-diagnostic examination, one should employ a printed chart, in which the nerves and muscles are conveniently arranged with spaces opposite for the insertion of the observations. This both saves time and prevents the accidental omission of important muscles from examination. A convenient form of chart is supplied by Messrs. Danielsson & Co.

CHAPTER X.

EXAMINATION OF THE EYE, EAR, THROAT, AND NOSE.*

SECTION I.—THE EYE.

NOTE first any obvious peculiarity about the eye. Observe whether there is any undue prominence of one or both eyes. Prominence of the eyes occurs in exophthalmic goitre. It is associated in that disease with the presence of what is known as *von Graefe's sign*. Ask the patient to look down. If von Graefe's sign is present, the upper lid seems to lag behind the eyeball in its descent, so that a large part of the upper portion of the sclerotic becomes visible. In paralysis of the cervical sympathetic the eyeball recedes so as to look more sunken than normal.

Note also whether the winking movements are increased or diminished in frequency. Infrequency of the movements constitutes *Stellwag's sign* of exophthalmic goitre.

The occurrence of squint, ptosis, retraction of the upper lid, and alterations in the pupil have already been considered. The presence of any inflammation along the margins of the lids (marginal blepharitis) should always be noted. It is often an indication of a strumous constitution. Next turn your attention to the **conjunctiva.** It may be necessary to examine the conjunctiva lining the eyelids. In order to do this in the case of the lower lid it is sufficient to depress the latter firmly with the thumb. To expose the inner surface of the upper lid, place the right thumb at the upper part of the upper lid and push upwards, so as to make the eyelashes stand out prominently. Grasp the lashes between the forefinger and thumb of the other hand, and evert the lid by rotating it round the thumb of the right hand.* Note the colour of the conjunctiva—whether it is pale, injected, or jaundiced. The method of detecting œdema of it has already been described. Look at the **cornea.** Note the presence of any ulceration or opacity of it. Small opacities are described as "*nebulæ,*" larger opacities are spoken of as "*leucomata.*" Try to make out whether the opacity is really on the surface of the cornea or deeper down in its substance. This can best be determined by looking along the surface of the cornea, as it were, and observing whether the light is reflected from it over the opaque spot, or whether it is dull. If the former be the case, the opacity is deep-seated; if the latter, it is superficial. Such superficial opacities point to former strumous ulceration. Deep-seated opacities are often due to congenital syphilis.

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* In this chapter only those methods of investigation are *described that* are required in ordinary medical cases.

The term *arcus senilis* is applied to a crescentic opacity which is sometimes observed towards the margin of the cornea. It usually appears at the upper part of the cornea first, and then gradually extends round. It occurs normally in old people, and is sometimes observed also in the eyes of younger persons who have sclerosed arterial walls and other signs of premature senility. True arcus senilis can be recognised by its leaving a small strip of clear cornea between the arc and the sclerotic; a crescentic opacity extending inwards from the sclerotic, which is sometimes met with, leaves no such clear strip. If arcus senilis be observed its presence should always be noted.

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* The following simple method of everting the upper eyelid, so as to permit of an inspection of the conjunctiva, is described by Lang in his book on "The Methodical Examination of the Eye":—"Whilst the patient, with head erect, turns his eyes down and looks towards the ground, the observer, who stands in front, places the end of his left forefinger on the right upper lid just above the tarsus, and his thumb on the lower lid just below the tarsus. By moving the forefinger a little upwards the margin of the upper lid is tilted forwards away from the globe; the thumb is now made to push the edge of the lower lid upwards into the space between the upper lid and the cornea. The lower lid thus acts the part of a wedge, and drives the upper lid forwards until its conjunctival surface is in contact with the thumb. When the whole thickness of the upper lid is between the finger and the thumb, the lower lid is released and the free edge of the tarsus is pushed upwards and backwards by the thumb whilst the forefinger presses its attached margin downwards and forwards; in this way the lid is everted. The right hand is employed to evert the left lid."

It is often of importance in medical cases to be able to say whether a patient is suffering from iritis or merely from conjunctivitis. In each case the eye looks red and injected, but the characters of the injection are different in the two conditions. They are contrasted in the following table:—

Conjunctival Injection.	*Ciliary Injection* (Iritis).
Colour, brick red.	Colour, pink.
Vessels very tortuous.	Vessels, straight.
Vessels can be moved on sclerotic injection, greatest on lids and in cul-de-sac, diminishes round cornea.	Vessels cannot be moved. Injection greatest round cornea, diminishes towards periphery.
Pressure on lid leaves no anæmia.	Pressure on lid leaves temporary anæmic spot.

In taking the case, note specially which of these sets of characters is present.

The **tension** of the eyeball should next be tested. This is done by placing the two forefingers on the upper part of the sclerotic outside the upper lid while the patient looks downwards, the other fingers resting on his forehead. Then try for fluctuation. The normal tension must be learnt *by practice,* and any increase or diminution of it noted. An increased tension contra-indicates the use of atropine. Having observed these different points, one should next proceed to what is termed **oblique focal illumination** of the eyeball. For this purpose it is best to have the patient in a dark room, a lamp being placed in front of him. By means of a convex lens—the ophthalmoscope lens does very well—the light is focussed on the surface of the eye. If necessary, one may hold another lens in the left hand, and so magnify the view. Study in this way the surface of the cornea. The nature of the opacities already referred to can now be observed more minutely. Look at the iris. The outline of the pupil, its contractility to light, the existence of synechiæ, and the presence or absence of hippus, can all be very well observed by this method. Note whether any opacity can be detected behind the pupil, and if so, try to estimate the depth at which it is situated.

Then proceed to **retinoscopy.** Use for the purpose a plane or slightly concave mirror, with an aperture in the centre. An ordinary ophthalmoscope mirror does very well, but it is preferable to use one of larger size, say about two inches in diameter. The patient should be in a dark room with a light just above his head, and it is well to have his eyes atropised. Sit fully a yard from him, and ask him to look far away over the top of your head. Then throw the light into his eye by means of the mirror. In a normal eye a red reflection from the retina will be seen through the pupil. If there be any opacity of the refractile media, the red reflection will be obscured. In this way cataract may be detected. When commencing it usually takes the form of opaque bands passing in towards the centre of the pupil like the spokes of a wheel. Then tilt the mirror, first upwards and downwards, then from side to side. As one does so, a black shadow is observed to flit across the pupil. Watch the edge of this shadow. From the direction in which it moves information is obtained as to the state of refraction of the eye. If the eye be emmetropic, or if it be hypermetropic, or has less than one dioptre of myopia, the edge of the shadow moves in the opposite direction to that in which the mirror is tilted if it be concave, but in the same direction to that in which it is tilted if the mirror be plane. In myopia of more than one dioptre the edge of the shadow moves in the same direction as a concave mirror, but in the opposite direction to one which is plane. In a normal eye the shadow moves very rapidly, and has a straight and sharply-defined edge.* The more abnormal the patient's refraction, the more slowly does the shadow move, and the more crescentic and the less well defined is its margin.

If the edge of the shadow moves differently in opposite meridians, the eye is **astigmatic.** If one meridian is normal the astigmatism is *simple*, and may either be of the myopic or of the hypermetropic variety, according to the nature of the refraction in the abnormal meridian. If both meridians are abnormal, the error of refraction being the same in kind in each, but unequal in degree, *compound astigmatism* is present. It may also be either of the myopic or of the hypermetropic variety, according to the nature of the refraction. If one meridian is myopic, the other hypermetropic, the condition is one of *mixed astigmatism.*

In *regular astigmatism* the directions of greatest and least refraction are at right angles to each other, and usually fall exactly in the vertical and horizontal meridians, the meridian of greatest curvature being most frequently the vertical. Sometimes, however, the directions of greatest and least refraction are in the oblique meridians.

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* The edge of the shadow is, of course, straight only when the source of light has a straight edge. This is so in an Argand burner, but not in an electric lamp.

In *irregular astigmatism* the directions of greatest and least refraction are not at right angles. This occurs comparatively rarely.

For the optical explanation of these facts, and for the more detailed description of their significance, the reader is referred to special works on ophthalmology. We would only remark here that retinoscopy affords the simplest and readiest method of arriving at an idea of the state of a patient's refraction.* In examining many medical cases such information is well worth obtaining, as errors of refraction have been found to be the reflex cause of many nervous symptoms, *e.g.* of headache, vomiting, etc.

The *fundus oculi* remains still to be examined, and for this one must have recourse to the use of the **ophthalmoscope.** Many patterns of this instrument are sold. The essential points are that there should be two mirrors—a larger one for use in the indirect method of examination, and a smaller angled one for direct examination of the fundus. It is also important to be able to bring a series of small lenses of different refraction behind the eyehole in the mirror.

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* Sometimes a patient comes before one wearing glasses, and it may be important to know what the refraction of these is. In order to discover this, hold the glass in front of the eye and look at an object through it. Then move the glass from side to side, and watch the object. If the latter seems to move in the opposite direction to the glass, the latter is convex; if in the same direction, it is concave.

The strength of the glass may be approximately determined by bringing the small lenses of the ophthalmoscope behind it, until one finds that which abolishes the apparent movement of the object looked at.

In order to tell whether the glass is spherical or cylindrical, look at a straight object, *e.g.* a window bar, through the glass, *and then slowly* twist the latter round. If the glass is cylindrical, *the object looked* at will appear to take up an oblique position. *Patients who use* cylindrical glasses are astigmatic.

There are two methods of using the ophthalmoscope—the indirect and the direct. We shall describe the former first.

1. **Indirect ophthalmoscopy.**—It is best to have the room darkened; at any rate, bright sunlight should be excluded. If the patient is in bed this may be effected by placing an umbrella over him. The patient (if he is able) sits in a chair with his head *slightly* inclined forwards, and the observer in another, about two feet from that of the patient, and directly opposite to the latter. A light—an ordinary oil lamp, or, preferably, an Argand burner—is placed close to the patient and on a level with his left shoulder. The observer should sit rather obliquely, his left shoulder being turned well round towards the patient. The ophthalmoscope is held in the right hand, the larger mirror being opposite the eye-hole. If the observer is not emmetropic, he corrects his own error of refraction by means of the small lenses behind the mirror; light is then reflected into the patient's eye from the lamp. A red reflection from the retina should fill up the entire pupil unless there be any opacity of the media, as already mentioned. No details of the fundus should yet be visible. If any blood-vessels be seen, one can be sure that the patient's refraction is abnormal. If these seem to move in the same direction as the head of the observer, the patient is hypermetropic; if in the opposite direction, he is myopic. Having got the fundus fully illuminated, one proceeds to interpose the convex lens of the ophthalmoscope. Hold it between the finger and thumb of the left hand, so that it rests opposite the joints between the first *and second* phalanges of the index. The finger should

not be at all flexed. The fore-arm should be kept vertical, the hand drooping from the wrist, and the little finger supported on the patient's forehead. This is the position demanding the least muscular effort, and therefore the least fatiguing to the observer. The ordinary ophthalmoscopic lens is of about + 15 dioptres strength, and therefore requires to be held about 2½ inches from the patient's eye.*

The exact point at which it should be held is arrived at by moving the lens backwards and forwards till a clear image is obtained.

A larger image of the fundus can be obtained by using a convex lens of about + 10 D (4 inches focus) and magnifying the image by means of a + 2 D lens placed behind the mirror. In many ways this is preferable to the ordinary method. Having got the fundus into focus, one wishes to look at three parts of it—(1) at the optic disc, (2) at the macular region, (3) at the periphery. In order to bring the disc into view, the patient must be made to turn his eye somewhat inwards. If the left eye is being examined, ask him to look at the tip of your left ear; if the right is being examined, ask him to look at the tip of the little finger of the right hand, which is stretched out for the purpose beyond the handle of the ophthalmoscope.

In order to see the macular region ask the patient to bring the eye slowly back from the above position towards the centre of your forehead. The macular region is reached at about two discs' breadth from the margin of the disc. Attempts to look straight at the macula in this way are sometimes baffled by the great contraction of the pupil which results, and by the reflection of light from the surface of the cornea. When this is the case it is better to ask the patient to look, not straight at the forehead, but a little to one side. The observer must then move his head till a view of the macular region is obtained.

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* A lens of 1 dioptre (1 D) strength has a focal distance of 1 metre (40 inches); a lens of 2 D has a focal distance of 20 inches, and so on. To signify a convex lens the sign + is used, thus: + 2 D means a convex lens of 2 dioptres; − 2 D means a concave lens of 2 dioptres. A lens which is curved equally in every direction—*i.e.* which is part of a sphere—is called *spherical*. It may be either convex or concave. A concave spherical lens of 1 dioptre strength is indicated thus: − 1 D spher.

A *cylindrical* lens is part of a cylinder, and is therefore curved in one direction only. The direction corresponding to the axis of the cylinder is uncurved, and is spoken of as the axis of the lens. A convex cylindrical lens of 1 dioptre strength is written + 1 D cyl. Cylindrical lenses are required in astigmatism.

The periphery of the fundus is seen by asking the patient to look first to his extreme right and left, then up to the ceiling, then downwards.

If, on gradually withdrawing the lens, the image of the fundus appears to become larger, there is *myopia* present. If it becomes smaller, there is *hypermetropia.* If it alters in one direction and not in others, there is *simple astigmatism;* if it alters in one direction more than others, the astigmatism is *compound.*

If, on moving the lens from side to side, one part of the fundus seems to move over the rest ("parallactic movement"), that part is at a higher level than its surroundings. Thus, if the disc be excavated, its margin will appear to move over the deeper part.

The examination of the fundus, and especially of its peripheral region, is greatly facilitated by a preliminary dilatation of the pupil. The best way of effecting this is to apply a 2% homatropine and cocaine ointment to the eye about half an hour before examining it. The effect may be afterwards counteracted by instilling a drop of eserine solution (1%).

If the patient be unable to leave bed, indirect ophthalmoscopy may easily be carried out by the above method, provided he is able to be propped up. If *that is impossible,* place the lamp on the pillow above his head, and carry out the rest of the procedure as above.

2. **Direct ophthalmoscopy.**—The patient is placed as before, but with the light a little behind and above the shoulder corresponding to the eye under examination. The observer sits quite close to the patient, so that his eye can be advanced to within two inches of that of the latter. In examining the left eye of the patient, use your own left eye, and for his right use your right. Tilt the patient's head and your own in opposite directions, so as to avoid breathing one another's breath. Arrange the ophthalmoscope with the small oblique mirror opposite the eye-hole, and its surface directed towards the light. The apex of the wedge formed by the tilt of the mirror should be directed towards the root of the observer's nose, when the instrument is held flat against his cheek. If there is difficulty in getting proper illumination of the fundus, move the source of light about until the bright red reflection is seen through the pupil. Ask the patient to look over your shoulder at a distant object, and try to relax your own accommodation entirely. This is the real point of difficulty in the direct method. As one is desirous of seeing clearly the fundus of the patient, and as that is so near, one almost instinctively accommodates his own eye for a near object. With practice, however, this difficulty can be overcome. It sometimes helps one to achieve the desired result if he tries to think in a dreamy way of some distant object, and to picture it to himself. If it is found impossible at first to relax one's accommodation completely, it may be nullified by the use of a — 2–3 D lens behind the mirror.

When the fundus has been brought into clear *focus, its different* regions must be systematically *studied, just* as in the use of the indirect method. In order to see the disc by the direct method, look backwards and inwards obliquely into the eye, telling the patient meanwhile to look straight in front of him.

In the case of a patient who is unable to rise from bed, the direct method may be applied in one of several ways.

(1) The observer kneels beside the bed at right angles to the patient, the light being placed on the pillow at the opposite side of the head of the latter. (2) The observer places himself at the top of the bed, so as to look down, as it were, on the patient's eyes, the light being placed on the opposite side of the head from the eye to be examined. (3) If the patient be a child, place him across the bed, and kneel at the patient's head, the light being held at the opposite side from the eye under examination.

The images furnished by the two methods of ophthalmoscopy differ. In the indirect method the image is inverted, so that what seems to be the upper part of the fundus is really the lower, and the inner (nasal) side appears to be the outer, and *vice versâ.* The image, however, embraces a large part of the fundus at one time, so that one gets, as it were, a bird's eye view of it. This method is, therefore, well suited for ordinary diagnostic purposes in medical cases.

The image obtained by the direct method is an upright image, and therefore the different parts of the fundus are seen in their proper positions. The image embraces only a small part of the fundus at one time, but gives a magnified view of it. It is, therefore, well suited for the minute study of pathological changes in the fundus.

The image obtained by the modification of the indirect method already described (*i.e.* by interposing a weak convex lens, and magnifying the image by a *+2 D lens* behind the mirror) is intermediate between the images obtained by the ordinary indirect and by the direct method. It is an inverted image, pretty highly magnified, and shows also a fairly large part of the fundus at one time.

We have said that the optic disc, the macular region, and the periphery of the fundus must be studied in detail in each case. We may now pass to a consideration of the special points to be taken note of in each of these regions.

1. Note as regards the **optic disc**—

(1) Its *shape*. Normally this is circular. Sometimes it is oval. If there be astigmatism present, the disc will appear to be oval, although it is really circular. This apparent oval shape may be distinguished from that which is real by moving the lens backwards and forwards. If the disc be really oval, it remains unaltered; if it is only apparently oval, its shape will be found to vary with the position of the lens.

(2) Its *colour*. The normal disc is of a rosy tint, but distinctly paler than the rest of the fundus. The nasal side is normally rather redder than the other.

In atrophy of the optic nerve, the disc becomes very pale, and may even be dead white or greyish in tint. In active hyperæmia of the disc its colour approaches in intensity to that of the rest of the fundus. Such active hyperæmia is often present in high degrees of hypermetropia. In passive hyperæmia of the disc the veins are alone affected (p. 512), and the general tint is not altered.

(3) The presence or absence of a *physiological pit*, and its size. Do not mistake the pallor produced by a very large pit for the pallor of optic atrophy.

(4) The character of the *vessels*. The arteries are *normally distinguished* from the veins by the following *characters*:—They are only $\frac{2}{3}$–$\frac{3}{4}$ the breadth of the

veins, and they are not so dark in colour They have a broader, better defined, and more continuous light stripe along their centres.

Normally, the arteries do not pulsate. They may be observed to do so in cases of aortic regurgitation, and in increased intra-ocular tension. The veins sometimes pulsate, even in normal eyes, either owing to the twisting of the arteries round them at points, or to high intra-ocular tension.

(5) The *edge of the disc.* It should be clear and well defined—especially at its outer side. As the vessels run across it, they should not be observed to tumble over at all. This "tumbling over," if present, is best evinced by the sudden disappearance of the central light stripe on the vessel.

(6) The *surroundings of the disc.* This part of the fundus should be carefully searched if one is on the outlook for the presence of hæmorrhages or tubercles, as both of these are more often encountered in the immediate neighbourhood of the disc than at other parts of the fundus.

Tubercles will be recognised as roundish, ill-defined, yellowish bodies, usually about half as large as the disc. *Hœmorrhages* occur as bright or dark-red blotches, with flame-like margins.

2. The **macular region** is situated, as we have said, about two discs' breadth from the outer edge of the disc. It is recognised by being rather darker in colour than the rest of the fundus, by being very devoid of blood-vessels, and frequently by being surrounded with a halo of reflected light, producing a shot-silk appearance. The macula itself is in the centre of the region, and is rather pale in colour, and often glitters somewhat.

Changes in the macular region are important, in that they interfere more with vision than similar changes in any other part of the fundus. In cases of albuminuric retinitis, a circle of white spots may often be observed arranged around the macula (p. 515).

3. **Periphery.**—Inspection of the periphery of the retina is important, as it is here that some changes—such, for example, as disseminated choroiditis and retinitis pigmentosa — are most early to be detected.

The following is a brief description of the chief changes met with in the fundus which are of importance from a medical point of view :—

Optic neuritis.—In medical cases this is usually bilateral (double optic neuritis). It begins as a mere passive congestion of the disc, with slight œdema. At this stage the veins are fuller than normal ; on indirect examination the edge of the disc seems clear enough, but on closer inspection by the direct method it is seen to be slightly fluffy-looking. The change in the edge of the disc usually begins at its upper and lower margins. These parts should therefore always be most carefully inspected.

As the process progresses, it passes into true neuritis, or papillitis. The disc is now distinctly swollen. This is best recognised by the fact that, on direct examination, one requires (provided his accommodation is fully relaxed) the aid of a convex lens behind the mirror in order to bring the vessels on the disc clearly into focus. The veins are still larger than before, and distinctly tortuous. Pathological tortuosity of the veins occurs at right angles to the plane of the retina. Tortuosity in the same plane as the retina may be quite normal. Often the veins can be observed to tumble, as it were, over the edge of the swollen disc. The arteries are smaller than normal, and may be partly obscured by the presence of exudation. The edge of the disc is no longer clear, even on indirect examination, but fades off into the surrounding retina. Small hæmorrhages may be observed on or near the disc.

It is often important to decide whether the papillitis is advancing or not. One ought not to form an opinion on this point unless he has already examined the disc on a previous occasion. The best criterion is the degree of swelling of the disc. In order to estimate this, use the direct method, and be sure that your own accommodation is thoroughly relaxed. Notice first whether the retina can be seen quite clearly without the aid of a lens. If the eye is emmetropic, one ought to be able to do so. If the refraction is abnormal, place behind the mirror the lens which is required to bring the vessels on the retina clearly into focus. Then look at the vessels on the disc. Owing to the swelling of the latter, the vessels are nearer the observer's eye than they should be, and a + lens must therefore be brought behind the mirror in order to enable one to focus them clearly. The strength of the lens required is the gauge of the amount of swelling which is present in the disc. Suppose, for example, that one requires to use + 1 D in order to focus the retina clearly (*i.e.* the patient has 1 D of hypermetropia), but that in order to focus the vessels on the disc one requires to make use of a + 6 D, then there is obviously + 5 D of swelling. Roughly speaking, every 3 D = 1 millimetre of swelling. In this way, one can estimate the amount of swelling from day to day, and so determine whether the neuritis is advancing or receding. The above method, it is obvious, requires a considerable amount of practice, and it is absolutely essential for success that the observer should be able thoroughly to relax his own accommodation.

Those who are unable thoroughly to relax their accommodation may use the following method instead of the above:—Bring behind the mirror the weakest

— or strongest + lens with which (*a*) the vessels on the fundus can still be clearly defined; do the same for (*b*) the vessels on the top of the disc. Then $\frac{b - a}{3}$ = the height of the disc in millimetres.

Optic neuritis is present in most cases of cerebral tumour at some period of the disease. It occurs also in about 50 per cent. of cases of tubercular meningitis, although it is often late in making its appearance. It is very uncommon to meet with it in the simple basal meningitis of infants, and in ordinary acute meningitis. It is also infrequent in cerebral abscess, and is not met with in cases of cerebral hæmorrhage, or thrombosis. On the other hand, it is not infrequent in other than intracranial diseases, and especially in Bright's disease (most commonly in the form which is associated with cirrhosis of the kidney and thickening of the blood-vessels). It is also met with in lead poisoning, and in some cases of anæmia.

Optic atrophy.—We have already mentioned that the most striking change in the fundus in this condition is the pallor of the disc and the smallness of the arteries on it. The atrophy may be (*a*) primary (*e.g.* in locomotor ataxy)—this is sometimes called simple atrophy; or (*b*) it may be post-neuritic atrophy; or (*c*) it may follow degenerative changes in the retina. It is not always easy to say from a mere inspection of the fundus which variety it is that one has to deal with, and the longer the process has gone on the more difficult does the diagnosis become. Look at the edges of the disc. If they are indistinct, especially if white streaks can be seen radiating out from them and passing along the vessels into the retina, then one can be pretty sure that the atrophy is not primary but is post-neuritic in origin. If the atrophy is secondary to changes in the retina the disc looks like a bit of dirty parchment, and pigmentary changes will be seen in the retina. In atrophy without previous optic neuritis the thinning of the nerve fibres renders very visible the fibres of the lamina cribosa, and the disc acquires a "mottled" appearance. The presence of such mottling should always be carefully noted.

Albuminuric retinitis.—This condition of the fundus is met with in some cases of Bright's disease, especially in the very chronic forms. The changes consist in (1) the presence of optic neuritis with marked fulness of the veins; (2) the occurrence of hæmorrhages on or near the disc; (3) the development of white shining spots around the disc at a distance of about three discs' breadth from it, and of similar but much smaller spots arranged in a stellate form around the macular region. Any one of these sets of changes may be present without the others. The presence of these changes should be looked for in every case of chronic renal disease.

Tubercle of the choroid and hæmorrhages in the retina have already been mentioned, and their commonest sites pointed out. Tubercles may be looked for in cases of suspected acute miliary tuberculosis and tubercular meningitis. As they only appear very late in the affection, however, they are rather of pathological interest than of diagnostic importance.

Retinal hæmorrhages may occur in any form of profound anæmia, but especially in pernicious anæmia and leucocythæmia. They also occur in purpura.

Embolism of the central artery of the retina may occur in cases of endocarditis. The appearance of the fundus is characteristic. Look at the macular region especially. There is present at it a peculiar round cherry-red spot. The disc itself is pale and its arteries are empty. The retina as a whole is somewhat milky-looking from the presence of œdema.

Disseminated choroiditis.—This is often an important sign of previous syphilis. The changes observed are the presence of small white patches of various shapes and sizes, and disturbance of the pigment around them. These can be recognised as being situated in the choroid by the fact that the retinal vessels pass over them. In congenital syphilitic choroiditis there is often a marked heaping up of pigment around the patches, which are mostly circular in shape.

SECTION II.—THE EAR.

Examine first the **external ear.** Note any peculiarity of shape, observe the presence of any tophi, tumour, or swelling, or the existence of any skin eruption upon it. Observe whether there is any discharge from the meatus, and if so, make a note of its character and odour. Note also whether there is any redness, tenderness, or swelling over the mastoid.

The **meatus and membrane** must next be inspected. Daylight is best for the purpose. Place the patient with the ear to be examined turned away from the light. Use a slightly concave mirror of about three inches diameter and a focal distance of six inches, with an elliptical slit in the centre. In order to catch the light properly the surface of the mirror should be turned slightly upwards. By this means the external auditory meatus can be inspected. Note the presence of any foreign body, of an accumulation of wax, of any eczematous eruption or furuncles, or any other abnormality, as the presence of such may contra-indicate the use of the speculum.

Various forms of aspergillus may grow in the external auditory meatus and set up an otitis. The colour of the inspissated discharge depends upon that of the aspergillus, being black where A. niger is the fungus. The structure of the growth can be seen if some of the *débris* is mixed with a little liquor potassæ and glycerine and examined under the microscope.

In order to see the membrane, **a speculum** must be employed. A metal speculum is best, unless one is wishing to make use of caustic applications, in which case it is better to use a vulcanite instrument. Choose a size of speculum appropriate to the ear under examination, warm it slightly, and introduce it so that the long diameter of its smaller end is placed almost vertically, but with a slight inclination from above downwards and forwards. Take care not to introduce it too far—not beyond the cartilaginous part of the meatus. There is, of course, no use in attempting to dilate the osseous part. Having introduced the speculum, hold it in position by means of the forefinger and thumb of one hand, while the pinna is grasped between the ring and middle finger of the same hand, the mirror being held in the other. The pinna should be pulled gently upwards and backwards, so as to straighten the meatus as much as possible. The mirror is held with its surface looking slightly upwards as before, and the membrane can then usually be seen on looking down the speculum. If the view be obstructed by the presence of impacted wax, the latter should be removed by being first softened with warm almond or olive oil or soda solution (two teaspoonfuls of the bicarbonate to one pint), and then syringed out.

The student will find that it is not very easy at first to hold the speculum properly and at the same time to pull the pinna upwards and backwards. It is therefore better, if one can, to have the mirror attached to the forehead by means of a spectacle frame. This leaves both hands free to manipulate the ear and speculum, or to make applications by means of the probe, etc.

If an ear mirror be not at hand, the large mirror of an ophthalmoscope can be made to serve the purpose. The lens of the ophthalmoscope can also be held close to the speculum, and so a magnified view of the membrane is obtained. This is especially serviceable in examining the ears of children.

A better magnified view of the membrane can be obtained by aid of the **auriscope**.* This instrument consists of a plated German silver cylinder, connected at one end with an ear speculum, and at the other with an adjustable magnifying lens. Light is admitted through a funnel-shaped tube in the side of the instrument, and is reflected on to the membrane by means of an oblique mirror. Artificial light and a bull's-eye should be used. The patient is seated with the light on a level with his ear, and the bull's-eye against the end of the funnel. The auricle is drawn upwards and backwards with the left hand and the speculum gently introduced with the right. It is best to use the largest speculum one can.

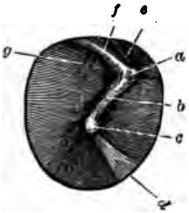

Fig. 140.—Outer aspect of right tympanic membrane—double the natural size. (*After Barr.*)

a, short process of malleus; *b*, handle of malleus: *c*, umbo; *d*, cone of light; *e*, membrana flaccida; *f*, posterior fold; *g*, long process of incus seen through the membrane.

The first thing noticeable about the **normal membrane** (Fig. 140) is its bluish-grey colour and translucency. A small white knuckle-like prominence may be observed towards the middle of its upper part. That is the short process of the malleus. Passing downwards and backwards from it may be noticed the long process of the malleus, which ends in the umbo near the centre of the membrane. Passing forwards and backwards from the short process of the malleus one notices the anterior and posterior folds of the membrane. A triangular light portion of the membrane usually catches the eye, the apex of which meets the lower end of the handle of the malleus at an obtuse angle which opens forwards. This bright spot is due to the reflection of light. Its presence may usually be accepted as a proof of a healthy state of the membrane. Immediately above the short process of the malleus a notch may be observed in the ring of bone which bounds the tympanic membrane. The part of the membrane which fills in this notch is called the membrana flaccida, or Shrapnell's membrane. It would be beyond the scope of this work to enter into a description of the various abnormal appearances which may be met with in the tympanic membrane. For the purpose of describing the situation of any abnormality which may be observed, it is customary to divide the membrane into an anterior part, which is in front of the handle of the malleus, and a posterior part, which lies behind it. Each of these is then divided by an imaginary line drawn through the tip of the handle into a superior and an inferior portion. Four quadrants are thus obtained, and in making notes one should say that a perforation (*e.g.*) is seen in the anterior superior quadrant, and so on.

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* *The best form* is Hovell's modification of Brunton's auriscope, *made by* Messrs. Mayer and Meltzer.

Inflation of the middle ear.—It is sometimes desirable *to inflate the middle ear with air.* This is best effected by aid of a Politzer's bag. The bag should have a piece of rubber tubing, about one inch and a half in length, attached to its nozzle. Give the patient a mouthful of water, and bid him keep it in his mouth until he is told to swallow it. Introduce the rubber tubing into the lower part of one nostril, pinch firmly the other nostril and the *upper part of* the one into which the tubing has been, introduced, and, holding the bag in the palm of the right hand, tell the patient to swallow. The moment you observe the larynx rising, squeeze the bag firmly, and the air will enter the middle ear.

To ensure the entry of the air into the ear, or to direct it into one ear only, the **passage of the Eustachian catheter** may be necessary. Hold the instrument lightly near the broad end with the thumb and two fingers of the right hand. With the thumb of the other hand push the point of the patient's nose gently upwards. Pass the end of the catheter into the nostril with the curve of the instrument looking downwards, and the handle somewhat lower than the point. Pass it backwards along the floor of the inferior meatus, and as soon as the curved part has entered the nostril raise the handle of the instrument until it is level, and continue to push it backwards until it comes in contact with the posterior wall of the pharynx. Then rotate the instrument till its point is directed towards the middle line, and withdraw it until the curve hooks against the posterior end of the nasal septum. Then rotate the instrument, so that the point sweeps downwards and then upwards and outwards, the handle being kept pressed towards the nasal septum, and stop when the ring of the instrument is directed towards the outer canthus of the eye of the same side. The point of the instrument can then usually be felt to be arrested by the cartilaginous rim of the tube. The nozzle of a Politzer's bag may then be introduced into the outer end of the instrument, and the inflation accomplished.

If one end of a rubber tube, with an ear-piece at each extremity, be inserted into the ear of the patient, and the other end into that of the observer, *the latter* can hear the sound which the air makes as it *impinges* against the membrane. If a whistling sound be heard, it indicates the existence of a dry perforation. A bubbling sound shows the presence of fluid.

SECTION III.—THE THROAT.

The methods of examining the fauces and the pharynx have already been considered (p. 49). In order to obtain a view of the larynx, one must have recourse to laryngoscopy. In performing **laryngoscopy,** the patient and observer should be seated opposite to one another in a darkened room, and about a foot apart. A light should be placed a little to the right (or left) of the patient's head and on a level with his mouth. An ordinary lamp will serve, but it is better to have an Argand burner, and it is also an advantage to have the light fitted with a bull's-eye condenser. The observer adjusts the reflector to his head by means of a forehead band or spectacle frame. If the former be used, the two knobs on the band should go against the root of the nose. It is then rotated on its ball and socket joint until the hole in the centre is directly opposite the right eye. This is ascertained by closing the left eye and observing whether one has a clear view through the aperture. One can also arrange the reflector so that it is in the centre of the forehead, and one then looks under its lower edge. This requires a little practice, but has the advantage of allowing one to make use of both eyes. It is also of advantage in the former method to have the aperture in the centre of the reflector in the form of an elongated slit rather than of a round hole, as a better view is thus obtained.

The observer should next manipulate the reflector with both hands until the light is directed on to the patient's mouth. He then selects a mirror, and warms it face downwards over the lamp until the moisture, which at first condenses on the surface, has all dried off. He should also touch his cheek with the back of the mirror before inserting it, in order to make sure that it is not too hot. Having warmed the mirror, he should hold it in such a way that it can be readily introduced and manipulated. On the whole, it is more convenient to hold the mirror like a pen than in any other way. It should also be held rather short, so that the hand of the observer can be steadied by resting the little finger on the patient's cheek.

The mirror being ready, the patient is told to crane out his neck a little, and to open his mouth and put out his tongue. The observer then throws a clean dry cloth over the anterior part of the latter, and grasps it firmly but gently between the forefinger and thumb of the left hand. It must be held firmly, but without any squeezing, and should then be, as it were, rolled out, as if round an imaginary axis situated near the hyoid. This manœuvre has the advantage of causing a better elevation of the epiglottis, whilst it prevents any risk of injuring the tongue against the lower incisor teeth. Before introducing the mirror make sure that the light from the reflector is concentrated on the back of the patient's throat. This having been ascertained, the mirror should next be introduced with its surface turned almost directly downwards, and passed rapidly back, care being taken to avoid touching either the tongue or the palate. The patient should be told to be sure to breathe regularly and through the nose. This serves to engross his attention. As the soft palate rises during an inspiration, the back of the mirror is placed gently against it, opposite the base of the uvula. The soft palate is then gently pushed upwards and backwards, and the handle of the instrument lowered or raised until the back of the epiglottis comes into view. The patient is then told

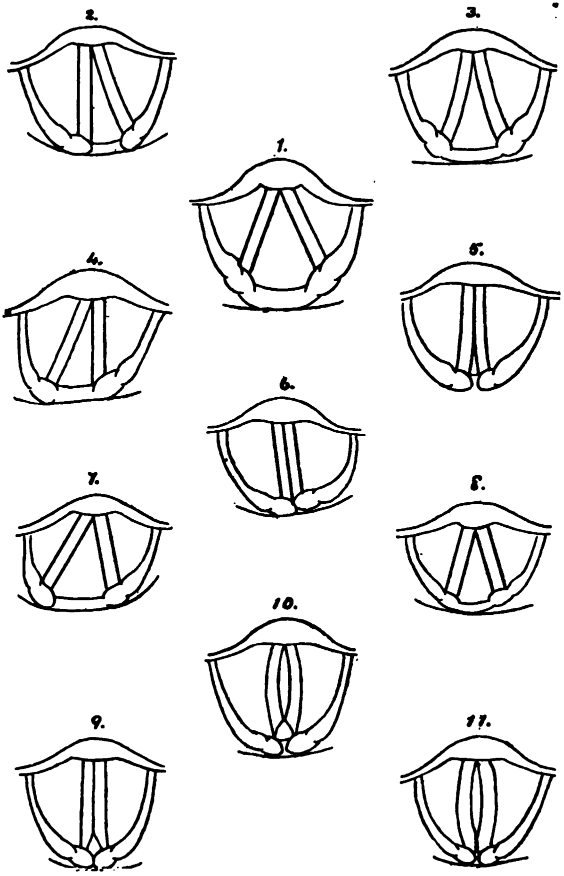

Fig. 141.—Diagram of Larynx.

1, normal larynx, respiration; PARALYSES:—2, adductor, left, phonation; 3, adductor, right and left, phonation; 4, abductor, left, respiration; 5, abductor, right and left, respiration; 6 recurrent, left, phonation; 7, recurrent, left, respiration; 8, recurrent, right and left, respiration and phonation; 9, arytenoid, phonation; 10, arytenoid and thyro-arytenoids, right and left phonation; 11, thyro-arytenoids, right and left, phonation. All drawn as seen in the mirror, *i.e.*, patient's left hand to observer's right.

to say "Eh," and that usually causes the vocal cords to become visible.

If the reflex excitability of the patient's pharynx be very great, so that any attempt to introduce the mirror induces retching, the application of a 10 per cent. solution of cocaine previous to beginning the examination will be found of great assistance.

It must be remembered also that one sometimes meets with a patient whose larynx baffles all attempts at inspection, owing to the position and shape of the epiglottis.

In studying the **view obtained,** the true cords usually first attract attention owing to their gleaming white colour. They should move freely on phonation. Tracing them forwards, they are seen to converge and disappear behind the cushion of the epiglottis. Posteriorly they diverge and terminate in knob-like prominences, which are the apices of the arytenoid cartilages. Immediately external to each of these is a smaller knob—the cartilage of Wrisberg. Passing backwards from each side of the epiglottis to the arytenoid cartilages are seen the aryepiglottic folds. In favourable circumstances the observer can see down the trachea, and even as far as its bifurcation. This is facilitated by the observer placing himself at a lower level than the patient, and holding the mirror in such a way that its surface looks almost directly downwards.

In observing any abnormal condition of the larynx, the chief points to be attended to are—

1. The colour of the cords and mucous membrane;
2. The presence of any swelling or ulceration;
3. The mobility of the vocal cords.

1. The normal colour of the vocal cords is a *pearly white*. In laryngitis they become red. Any *increase* or diminution in the redness of the laryngeal

mucous membrane should be noted. In tubercular affections the mucous membrane is abnormally pale; in syphilitic conditions it is unusually red.

2. The position and character of the swelling should be noted. Tumefaction of the aryepiglottic folds and of the epiglottis should be looked for in suspected laryngeal phthisis. Tumours of various sorts on the true cords are occasionally met with. As regards ulcers, note their number, their position, and the character of their floor. Tubercular ulcers are usually multiple, and are met with very frequently on the interarytenoid fold. Syphilitic ulcers are usually single, and have a yellow sloughy floor.

3. Observe whether the cords come together normally on phonation and open widely during inspiration. In **adductor paralysis** the affected cord fails to move inwards on phonation, or the cord makes a sudden movement inwards and then goes back, the position being unsustained (Fig. 141—*2*, *3*). In **abductor paralysis** the cord looks normal on phonation, but fails to move outwards again on inspiration (Fig. 141—*4*, *5*).

In paralysis of both abductors and adductors (paralysis of the whole recurrent laryngeal nerve, or "**recurrent paralysis**") the cord is fixed in the cadaveric position—*i.e.* midway between complete adduction and abduction. This is much more common on the left than on the right side, owing to the greater liability of the left recurrent laryngeal nerve to be pressed upon by aneurysms (Fig. 141—*6*, *7*, *8*).

Adductor paralysis is usually the result of functional disease. Abductor paralysis, on the other hand, is the form of paralysis characteristic of an organic lesion of the nervous system. Bilateral adductor paralysis or paresis is the cause of the condition known as hysterical aphonia.

If on phonation the cords come together incompletely, leaving an elliptical space between them, there is **paralysis of the internal thyro-arytenoids** present (Fig. 141—*11*). If the anterior two-thirds of the cords come together, but leave a triangular cleft behind, the **interarytenoid muscle** is affected (Fig. 141—*9*). For further details regarding these forms of paralysis special works must be consulted.

SECTION IV.—THE NOSE.

Anterior rhinoscopy.—The position of the patient and of the observer, and the arrangement of the light and reflector, are the same as for laryngoscopy.

The anterior nares should first be inspected without the aid of a speculum. Tilt the tip of the nose upwards with the finger, and note the presence of any eczematous or ulcerated condition of the mucous membrane or skin. Observe whether any dried secretion or blood can be seen. Look for any swelling, ulceration, or perforation of the cartilaginous part of the septum.

Having noted these points, take a solid two-bladed speculum, warm, and introduce it. Hold it in position with the left hand, and gently screw the blades apart with the right. The first object to be observed is usually the anterior end of the inferior turbinated body. Note whether it is larger than normal or not. If it be enlarged, touch it with a probe so as to ascertain whether the enlargement be osseous or due to swelling of the mucous membrane merely. Then depress the patient's chin somewhat so as to bring the inferior meatus into view; then ask him to hold his head a little back so as to obtain a view of the middle meatus and middle turbinated body. The latter is considerably lighter in colour than the inferior turbinated. The superior meatus can never be seen, and the superior turbinated only very rarely.

The presence of polypi should be specially looked for in these parts. Their recognition is facilitated by the use of the probe. Lastly, turn the patient's head a little, so that the septum can be inspected. Note any deviation of it, or the presence of any prominence or spine, or the existence of any ulceration or perforation.

Swelling of the inferior turbinated body sometimes obstructs the view of the rest of the nasal cavity. The application of a little 10 per cent. cocaine on a pledget of wool will usually cause the swelling to disappear.

Posterior rhinoscopy. — This is the only method of obtaining a view of the posterior nares. In carrying it out, the position of the observer, the patient, the reflector, and the light should be the same as for laryngoscopy. The patient, however, should have the chin rather depressed, the neck not being craned out as in the examination of the larynx.

Select the smallest laryngeal mirror, warm it, and ask the patient to open his mouth. It is sometimes an advantage to have the shaft of the mirror bent back a little about 1½ in. above the reflecting surface. Introduce a right-angled tongue depressor, and hold down the tongue with the left hand. Take the mirror in the right hand, and pass it in with the surface looking upwards. Introduce it behind the soft palate, passing along between the uvula and the left anterior pillar of the fauces. Then turn the mirror a little, so that its surface looks upwards and forwards. The posterior nares will then come into view. What one usually sees first is the posterior end of the nasal septum. It looks narrow, sharp, and pale in colour below, but expands a little and looks reddish above; a slight cushion-like swelling can also be often seen about the middle of it on each side (Fig. 142).

On the outer wall the posterior end of the middle turbinated bone can usually be easily seen as a large bluish-red swelling. Above it, one can see the superior meatus and the end of the superior turbinated bone;

below it is the middle meatus and the upper part of the inferior turbinate. Observe the presence of any increase in size of any of these objects. Note also the general character and colour of the mucous membrane, or whether any mucus or pus can be seen adhering to it. Next turn the mirror a little upwards and to one side, keeping it rather low down, and with its back against the tonsil, and look for the cushion of the opposite Eustachian tube, which can usually be made out. It forms a bright red rounded projection, bounding a depression which leads to the orifice of the Eustachian tube. Observe whether there is any secretion at the mouth of the latter, or any adenoid swelling of the mucous membrane.

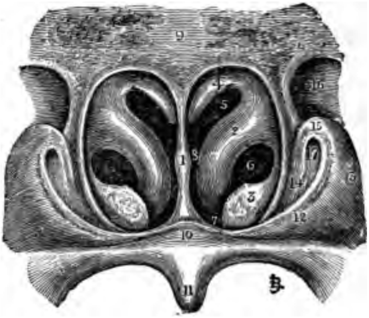

Fig. 142.—Posterior nares and surrounding parts.

1, septum; 2, middle turbinated bone; 3, inferior turbinated bone; 4, superior turbinated bone; 5, superior meatus; 6, middle meatus; 7, inferior meatus; 8, main passage of nostrils; 9, vault of pharynx; 10, cushion of soft palate; 11, posterior surface of uvula; 12, ridge formed by levator palati; 13, salpingo-pharyngeal fold; 14, salpingo-palatine fold; 15, Eustachian cushion 16, fossa of Rosenmüller; 17, Eustachian orifice.

Lastly turn the surface of the mirror upwards, and examine the vault of the naso-pharynx, noting especially the presence of any adenoid swelling or tumour in that region. Normally the roof should present an appearance not unlike that of the surface of the tonsil (Luschka's, or the pharyngeal tonsil).

Posterior rhinoscopy is often very difficult to perform. The difficulty may be due to there being very little room between the posterior wall of the pharynx and the soft palate. If this cannot be overcome by inducing the patient to breathe quietly through the nose, or to sniff, it may be necessary to introduce a palate retractor, but for a description of this process special works must be consulted. Even after the mirror is properly introduced, it is not always easy to recognise what one sees. This is largely due to the fact that only a small portion is seen at one time, and the mirror has to be turned about till every part has been viewed separately. Experience alone can overcome these difficulties.

CHAPTER XI.

LOCOMOTORY SYSTEM.

(*Bones, Joints, Gait.*)

THE locomotory system includes the muscles, bones and joints. The examination of the muscles is mos conveniently considered along with that of the nervou system (Chap. IX.). There remain for consideratio the bones and joints.

BONES AND JOINTS.

In examining the long bones of the limbs, look (1 at the condition of the shaft, (2) at the articular ends

As regards the **shaft,** note any distension or bending of the bone and any signs of a former fracture Then pass the hand along the bone, noting the presence of any tenderness or thickening of it. Thickening is most likely to be detected on the expose surfaces of bones, *e.g.* over the anterior surface of th tibia and at the lower ends of the radius and ulna Such thickening often affords valuable evidence of ol periostitis, especially of the syphilitic form.

As regards the **ends of the bones,** note th presence of any general enlargement, such as occur in rickets, or of any nodulation at the margins, suc as one finds in rheumatoid arthritis.

In examining a **joint,** note first the points whic can be made out by simple inspection. Observe th position in which the patient keeps the joint; not any alterations in its contour, such as local or genera swelling, and the presence or absence of any rednes Then pass to palpation, noting whether or not the is any increase of local heat in the joint, whether i is tender to the touch, and whether one can make ou the presence of any fluctuation in the joint cavit

Then try to move the joint, observing the degree of mobility in each direction, and whether or not attempts at movement produce pain. If the joint is movable, note whether any sensation of grating is produced on movement. If the mobility is limited in one or every direction, try to form a conclusion as to the cause of the limitation, and especially whether it is due to changes in the components of the joint itself, *e.g.* contraction of ligaments, or fibrous or bony anchylosis, or whether it is due to changes in the structures surrounding the joint, *e.g.* shortening of tendons. Next turn your attention to the synovial membrane. Try to make out whether there is any thickening or bogginess of it. Lastly, examine the articular surfaces of the bones, moving the joint (if possible) so that the edges of the articular surfaces can be examined with the fingers. Note the existence of any irregularity or thickening of these, and the presence of any outgrowth or "lipping" of them.

The **vertebral column and skull** demand special attention. Observe in the former the presence of any local projection of the vertebral spines. If such there be, state which are the vertebræ involved, and at which the projection is most prominent. In counting the vertebræ for this or any other purpose, one can take as landmarks either the spine of the vertebra prominens or the last rib, tracing the latter back to the twelfth dorsal vertebra.

In many cases, however, the last rib cannot be distinctly felt. It is therefore rather untrustworthy as a guide.

Note also the presence of any curvature of the spinal column as a whole, or of one part of it, distinguishing carefully such general curvature from the local projections above referred to.

The curvature may be in an anterior or a posterior *direction, or laterally.* Anterior curvature is termed *lordosis*, and is commonest in the lumbar region. General posterior curvature is spoken of as *kyphosis*. It occurs most typically in the dorsal region in old persons, and must be distinguished from the localised angular curvature of spinal caries. Lateral curvature is termed *scoliosis*, and may be towards either the right or the left side. It is always accompanied by a rotation of the bodies of the vertebræ in such a way that the spines come to point towards the concavity of the curve. An absence of the normal curves of the spine may be an indication of commencing vertebral disease.

Ask the patient to stoop down, and notice the degree of mobility of the vertebral column, and the occurrence of any pain during stooping, noting the exact site of the latter, if present. Then pass the hand down the vertebral column, and observe whether any tender spots can be made out. Such tender spots are not unfrequently met with in hysteria and in cases of irritation of the posterior nerve roots. Their presence can often be more easily elicited by drawing a sponge wrung out of hot water down the vertebral column; the patient complains of pain whenever the hyperæsthetic area is reached. To elicit more deep-seated tenderness of the vertebræ, it may be necessary to "punch" the spines gently with the fist from above downwards, observing the point at which the patient complains of pain and verifying the observation by repeating the process from below upwards.*

In studying the **skull**, note first its **size**. For this purpose it may be necessary to take **measurements**. This should be done in three directions: (1) antero-posteriorly from the root of the nose to the external occipital protuberance; (2) circumferentially at the level of a line drawn horizontally round the skull from the supra-orbital ridges in front to the external occipital protuberance behind; (3) coronally from one auditory meatus to the other. If the skull is abnormally small, the patient is microcephalic. This is frequent in some forms of idiocy. Abnormal enlargements of the skull occur in hydrocephalus, in ostitis deformans, and in acromegaly.

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* A better method of eliciting deep-seated tenderness of the spine, *e.g.* in early caries, is to press upon the top of the patient's head or upon the shoulders whilst he is in the erect position.

Next observe the **shape** of the skull. Is it of the dolichocephalic (long-headed), or the brachycephalic (bullet-headed) type? Are the two sides of the head symmetrical? Certain well-recognised types of abnormal skull are met with. In *hydrocephalus*, the skull tends to assume a globular form. The forehead is overhanging and the eyes are pushed down so that the upper part of the sclerotic is exposed. The lateral aspects of the skull (above the ears) project outwards. If the patient is a child, as is usually the case, the fontanelle is wide and bulging, and often fluctuates very distinctly. The sutures may be opened up, and imperfectly ossified areas (craniotabes) may be detected in the bones. In *rickets* the skull tends to be square or oblong and box-shaped. The frontal and parietal bones often show central thickening ("bossing"). The forehead, however, does not overhang, nor are the eyes depressed, and although the fontanelle is usually widely open, it does not bulge as it does in hydrocephalus, nor are the sutures opened up. In *congenital syphilis* the forehead is vertical, the frontal eminences are often exaggerated, and the bridge of the nose is depressed.

Having noted the general shape of the skull, ask the patient to open his mouth, so that one may see the **hard palate.** Observe its width and degree of arching. A high, much-arched, and narrow palate is often one of the minor signs of mental deficiency.

Next proceed to the **palpation of the skull.** Note first the thickness of the scalp, and whether it moves freely, as it ought, upon the subjacent bone. Atrophy and adherence of the scalp are apt to be associated with skin diseases in this region, and are often a bar to successful treatment. Observe the presence or absence of inequalities in the bones, such as may indicate the site of former injury or fracture. If a swelling be detected, pay special attention to its margins, noting whether a hard rim can be made out and whether or not the rim disappears on firm pressure steadily applied by the finger for a minute or two. In blood extravasations the rim disappears, in a depressed fracture it persists. Note also whether the swelling can be moved as a whole upon the skull or not. If the patient be a child, note the condition of the fontanelles and sutures, and look for the presence of unossified areas in the bones (craniotabes). The best place to look for these is in the neighbourhood of the lambdoidal suture. They feel like little spots which are covered only by parchment. Observe the presence of any tender spots or areas on the skull. For this purpose it may be necessary to tap the skull gently all over with the forefinger. If tenderness is detected, note carefully its maximum point. Such tender points are sometimes met with in inflammatory affections of the cranial bones or membranes, and in cases of superficially situated intracranial tumours, but they may also be present in neuralgic affections of the scalp.

THE GAIT.

The character of a patient's gait is often an important indication of the nature of the affection from which he is suffering. It is specially important in cases of nervous disease.

In studying the gait, it is well, if possible, to have the legs fully exposed. For this purpose the patient should have on only a night-shirt or dressing-gown, which should be brought through between the legs from behind, and pinned up in front. The feet should be bare. The patient is then told to walk away from the observer, to turn round at a given point, and then to come towards him again.

If it be desirable to obtain a permanent record of the patient's footprints, one can have recourse to photography. Put on the feet a pair of woollen socks, and dip them in rather thick whiting. Then ask the patient to walk along a smooth floor. The marks left by the feet can then be photographed.

In studying the gait, the **points to be noted** are :—(1) Can the patient walk at all? This being decided, one has to ask oneself—(2) Does he pursue a straight line, or does he tend to deviate to one side or the other, or to both alternately? To bring out this point, it is well to ask the patient to walk along a straight line—*e.g.* a crack in the floor. (3) Does he tend to fall, and, if so, in what direction? These questions being settled, the next point to be decided is whether the gait conforms to any of the well-recognised abnormal types. Before one tries to make up one's mind in this matter, however, it is well to be quite sure that the peculiarity in the patient's gait is not due to some surgical cause or to local disease of a joint—*e.g.* rheumatoid arthritis of the hip. For example, we have known the peculiar gait which is affected by patients with congenital double dislocation of the hip to be mistaken for the result of weakness of the muscles of the back, and treated by massage and electricity. A previous examination of the bones and joints will eliminate such sources of fallacy.

The three chief types of abnormal gait due to nervous affections are:—

1. The spastic;
2. The ataxic;
3. The reeling.

In taking a patient's case, it is usually sufficient to state that the gait belongs to one or other of these types, or is a combination of one or more of them. The chief peculiarities of each variety are as follows:—

1. The **spastic** may be described as a "*sticky*" gait. The patient has difficulty in bending his knees, and drags his feet along as if they were glued to the floor, the toes scraping the ground at each step. The foot is raised from the ground by tilting the pelvis, and the leg is then swung forwards, so that the foot tends to describe an arc.

This gait is seen most characteristically in patients with lateral sclerosis of the cord. The **hemiplegic gait** is essentially a spastic gait in which only one leg is affected.

2. The **ataxic** may be described as a "*stamping*" gait. The patient raises his feet very suddenly, often abnormally high, and then jerks them forward, bringing them to the ground again with a stamp, and often heel first. He seems to exhibit, also, an indefiniteness of purpose in the place chosen to put down the foot, and the feet while in the air do not move in one plane, but are waved about, as it were, before being set down. By adopting a "broad base," the patient tries to counteract the unsteadying effects of his style of progression. This gait is best seen in cases of locomotor ataxy.

3. The **reeling** gait may be described as a "*drunken*" gait, and, therefore, requires no further description. It will be observed that patients with *this gait* walk "on a broad base," the feet being *planted* widely apart. It is important to notice

whether supporting the patient by his axillæ abolishes the reeling tendency. In some cases of cerebellar disease, such support has been observed to abolish the patient's vertigo for the time during which he is supported.

This gait occurs most typically in cases of cerebellar lesion. It is, therefore, sometimes referred to as a "cerebellar gait."

Some rarer varieties of abnormal gait may be briefly referred to. These are :—

The "**festinant gait.**" This is the form of gait met with in typical cases of paralysis agitans. The patient is bent forwards, and advances with rapid short shuffling steps, so that, as has been said, "he looks as if he were trying to catch his centre of gravity." In some cases, if one suddenly pulls the patient backwards, he begins to walk backwards, and is unable to stop himself, though he is leaning forwards all the time. This peculiar phenomenon is spoken of as "*retropulsion.*"

The **waddling** or oscillating gait is like the gait of a duck. The body is usually tilted backwards, there being a degree of lordosis present; the feet are planted rather widely apart; and the body sways more or less from side to side as each step is taken. The heels and the toes tend to be brought down simultaneously. The chief peculiarities of this gait are due to a difficulty in maintaining the centre of gravity of the body owing to weakness of the muscles of the back. It is met with in pseudo-hypertrophic paralysis and in congenital dislocation of the hip.

The **high-stepping** or prancing gait is a device adopted by the patient to prevent his tripping from his toes catching the ground. It is, therefore, met with in cases where the toes tend to droop from weakness of the extensor muscles, *e.g.* in peripheral neuritis affecting the anterior tibial nerve. The name sufficiently describes its characters.

CHAPTER XII.

THE CLINICAL EXAMINATION OF CHILDREN.

THE clinical examination of young children is a matter full of difficulty to the inexperienced. It has to be carried out, not merely without the help of the patient, but often in spite of his strenuous opposition. In this chapter we propose to point out the best methods of ascertaining the necessary facts, and also the chief points in which the child differs from the adult in a clinical sense.

The history of the patient and his illness must, of course, in the case of young children, be ascertained from the mother or friends. The best **scheme of interrogation** to employ will be found on p. 11. Whilst the history is being elicited opportunity may be taken to cultivate the friendship of the child or, at all events, to get him accustomed to one's presence. The history having been ascertained, one proceeds to an examination of the child. This requires gentleness and deliberation, combined with infinite patience and good temper. If one is at all hurried or rough, the child begins to cry at once, and the subsequent examination is rendered a thousandfold more difficult. We would emphasise the fact that it is almost impossible to be really systematic in one's examination of children. Certain things must always be looked for, but no definite order can be observed in looking for them. One has to seize the opportunity of ascertaining a fact as it presents itself, and a rigorous adherence to "systems" is often out of the question. In the first place, a number of points can be ascertained before the child is undressed. One can study the **facies** of the child, note its complexion, the colour of its lips, and whether or not the alæ nasi are acting. One should also at this period of the examination count the respiration and pulse-rate ; it is very important to get these noted while the child is still undisturbed.

The **respirations** can usually be counted by merely watching the movements of the child's abdomen, that being very much more affected by respiration in young children than the chest is. The normal rate of a newly-born child is 40 or so respirations per minute, by the second year they have fallen to 30 or so, at the fifth year they are about 25, and by fifteen they have sunk to 20. Much more important than the absolute number of respirations is the ratio of respiration to pulse. Normally this should be as 1 : 3½ or 4.

The **pulse** is best counted by allowing the mother to hold the child's hand in hers; the fingers of the physician are then quietly slipped over the mother's hand on to the child's wrist and the pulse counted. If the child has begun to cry, it is useless to take the pulse-rate, as it may be at least 20 beats above the normal rate. The pulse-rate at birth should be 130, by the second year it has reached 110, by the fifth 100, by the eighth 90, and by the twelfth 80; after this it gradually sinks to the normal adult rate. During sleep the pulse-rate always falls about 10-20 beats. As a matter of fact, the examination of the pulse in infancy is of comparatively little clinical value. It is of little use as an index of the vital powers, the fontanelle replacing it in that respect. The vessel being extremely small, the characters of the pulse-wave can hardly be ascertained; irregularity by itself is of comparatively little significance, being very common, even in healthy infants, and being almost the rule in sleep. A pulse which is continuously *slow* and irregular is, however, of great significance.

These preliminary facts having been noted, the child should be stripped and placed in a blanket on the knee of the mother or nurse; examination must then be proceeded with by the usual methods of inspection, palpation, auscultation and percussion. In the clinical investigation of children it must be noted that the two former methods are of much the greatest assistance.

It is well to begin by looking and feeling the child all over. One notes the general state of development and nutrition, the state of the skin, whether dry and fevered, or moist, and the presence or absence of any rash or skin eruptions, and whether or not the normal degree of elasticity is present. The shape of the chest and the degree of prominence of the abdomen should be noted, it being borne in mind that the rickety and pigeon-breasted types of chest are very common in diseased children, and that a rather protuberant abdomen is to be regarded as normal. The hand should then be lightly passed over the head. The state of the **anterior fontanelle** should first be investigated. The fontanelle closes normally between the fifteenth month and the second year. If it remains patent after the second year, it is often a sign of disease — most usually of rickets. Too early closure of the fontanelle occurs in some forms of microcephaly and idiocy.

The degree of tension of the fontanelle is of great importance. In health it pulsates distinctly, and is neither sunken nor unduly elevated. A depressed fontanelle is an important sign of exhaustion; a tense fontanelle indicates increased intracranial pressure. It must be borne in mind, of course, that the fontanelle is normally tense when the child is crying. The systolic bruit heard over the fontanelle is of no clinical importance.

The **shape of the head** and of its bones must *be investigated.* The development of "**bosses**"

("Parrot's nodes") on the frontal and parietal bones is a common occurrence in rickets, especially in syphilitic children. One should also look for evidence of craniotabes (in young babes) and of rheumatic nodules (in older children). The general shape of the head as a whole should always be noted; it may be box-shaped as in rickets, or globular as in hydrocephalus; it may be abnormally small or large, or it may be asymmetrical.

Passing from the head, one may examine the **long bones.** In children, this is of extreme importance; many of the commonest and most serious diseases of infancy affect the bones more prominently than any other part of the body. Look for thickening or tenderness along the shafts of the bones. This may be due to scurvy, to syphilitic or to suppurative periostitis, or to tumours. Examine carefully the **epiphyses.** In rickets these become enlarged. This is most easily seen where the ribs join their cartilages, the thickening there forming a row of bead-like prominences ("**rickety rosary**"). It is also easily seen at the wrists. The frequency of inflammatory affections of the epiphyses should also be borne in mind. The presence or absence of "**rheumatic nodules**" should also be noted. These are little fibrous bodies varying in size from that of a large pin's head to a pea, or even bigger. They occur not in the periosteum, but in the deep fascia, where it covers superficial bones, and also in the sheaths of tendons. They should be looked for especially over the olecranon and patella. They are usually movable, but not tender. If found, they are pathognomonic of rheumatism. The **vertebral column** should always be examined for signs of tubercular disease or curvature.

At this point, if not earlier, the child's **temperature** should be taken. In young children the thermometer should be inserted into the rectum, or placed in the groin or axilla; in older children it may be placed in the mouth. It should be remembered that the temperature in children is much more variable than in adults, and often goes up upon very little provocation.

One must now proceed to the examination of the **thorax and abdomen.** The front of the chest and the abdomen may be examined together, and either after or before the posterior aspect of the chest. The order adopted should be, firstly, inspection and palpation, then auscultation, and, last of all, percussion. Percussion is left to the last, owing to the fact that it frequently makes the child cry.

In **palpation,** be sure that the hand is quite warm; this is even more important in examining the child than in the case of an adult. In **auscultation** one should use either the immediate method—the ear being applied to the skin directly (this is only applicable in examining the back), or else one should use a binaural stethoscope.* The latter enables one to follow slight movements on the part of the child better than one can with a wooden instrment. If the chest-piece of the stethoscope is made of metal, remember to warm it before applying it to the chest. There is only one point to be observed in the **percussion** of a child, and that is, that the stroke should be *light.* This is not merely in order to avoid frightening the little patient, but also to escape the confusion which is apt to arise from the excessive resonance of the child's chest.

When the abdomen and front of the chest have been run over in this way, one should turn his attention to the posterior aspect of the lungs. For the examination of these, the child should not be laid on his face, as that interferes with respiration, and causes the abdominal viscera to push up the diaphragm, but he should be held against the mother's breast with his head looking over her shoulder. In this way the whole of the back of the chest can be gone over.

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* The chest-piece of the stethoscope should be short, for when *the child is* on his mother's knee there is often but little space *between him* and her.

Last, but by no means least, comes the examination of the **mouth and throat.** It is impossible to exaggerate the importance of systematically examining the mouth and throat in all cases of illness in children. At the same time, it is just this part of the clinical examination in which we are most likely to meet with opposition; and for that reason it is left to the last, as it may be necessary to employ coercion in order to get it carried out.

Begin by looking at the **tongue.** Sometimes the child will put out its tongue when asked. In little babies gentle pressure on the chin will often cause the mouth to be opened, when a view of the tongue can be obtained. Or if a drop of milk or a little sugar be placed just outside the lip, the child will often put out its tongue in order to lick it off. In more refractory children it may be necessary to push the lower lip over the teeth, and then to press the lip down against the lower incisors. The child then opens its mouth in order to avoid having its lip cut. With very obstinate children one may be obliged to compress the nostrils until the mouth is opened to get breath.

Once the child has been induced, either voluntarily or by aid of one of the above devices, to open its mouth, one should note the state of the **buccal mucous membrane,** remembering the frequency of thrush, stomatitis, and ulcerations in children. The number and character of the **teeth** should be observed (*see* also p. 46), and the finger should be run along the gum to feel for any teeth that may *be about to* come through. One must then proceed to an examination of the **throat.** The child should be wrapped in a towel to restrain the movements of its arms. The mother or nurse sits down opposite a good light and takes the child on her lap. Another assistant steadies the child's head from behind. The child having then been induced or compelled to open its mouth, one introduces a small-sized tongue depressor and holds down the tongue, thus exposing the pharynx. The finger will often serve instead of an instrument, and has the advantage of frightening the child less. Look for any enlargement of the tonsils, for any redness of the mucous membrane, and especially for the presence of any membranous patches on it.

Palpation of the pharynx must also be carried out in many cases. To do this one must stand behind the child, and when its mouth is open push in the cheek from one side between its molar teeth. This serves as a gag, and effectually prevents the child from attempting to bite. The forefinger is then passed to the back of the pharynx and up behind the soft palate. Note the presence of any adenoids, or any bulging into the posterior wall of the pharynx, which may be the indication of the presence of a retro-pharyngeal abscess.

We have now indicated the general routine method to be employed in examining a child, but there remain some special points which we have not yet taken up.[*] These we shall consider briefly under the different systems:—

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[*] The following summary of the chief facts to be noted in the general inspection and palpation of a child may be of service. It is taken from a scheme in use by Dr. John Thomson:—

Appearance (if healthy or otherwise)—nutrition and development—complexion (anæmia, cyanosis, jaundice, etc.)—state of skin (dryness, moisture, eruptions, desquamation, pigmentation, *œdema)—attitude*, expression, demeanour, temper.

Shape of head and state of its ossification (fontanelle, cranio-

1. **General condition.**—Special importance attaches to the regular weighing of children. Alterations in **weight** from time to time are of much help in prognosis and treatment. It should be remembered that a healthy child should weigh at birth about seven pounds. This should be doubled by the time the fifth month is reached, and trebled in the first year. By the sixth year it is again doubled, so that a healthy child of six should weigh about three stones. This is again doubled when the fourteenth year is reached.

Measurement of the head is often of importance. Two measurements are usually sufficient—a coronal measurement from one auditory meatus to the other, and a circumferential measurement at the level of the root of the nose and external occipital protuberance.*

2. **Alimentary system.**—Note that the **liver** is normally rather large in children, and usually reaches at least half an inch below the costal margin. Enlargement of the **spleen** is very frequent in infantile diseases. It is best made out by palpation, the hand being passed across the child's abdomen from right to left. By depressing the finger tips opposite the eleventh interspace the edge of the spleen, if the organ is enlarged, may be felt as it descends during inspiration.

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tabes)—facial irritability—hair—eyes, nose, and ears (formation of, and if any discharge from)—shape of thorax, abdomen, back, and limbs (especially the hands)—enlarged glands—evidence of rickets, syphilis, and tuberculosis.

Character of voice, cry, and cough—rate and character of respiration (if noisy, dyspnœic, or painful)—movements of alæ nasi—rate and character of pulse—temperature.

Palpation of abdomen (tenderness, resistance, fluid, size of liver and spleen, tumours, etc.).

* The circumference of the head at nine months should be about 17 in.; at twelve months, about 19 in.; at seven years, 20-21 in. *After three years of age a circumferential measurement of 19 in. is too small.*

Inspection of the **stools** should never be omitted. The healthy infant, on the breast or bottle only, has two or three stools daily. These should be of the colour and consistence of beaten-up eggs. Any alterations in frequency, colour, or consistence, or the presence of worms, should be carefully noted.

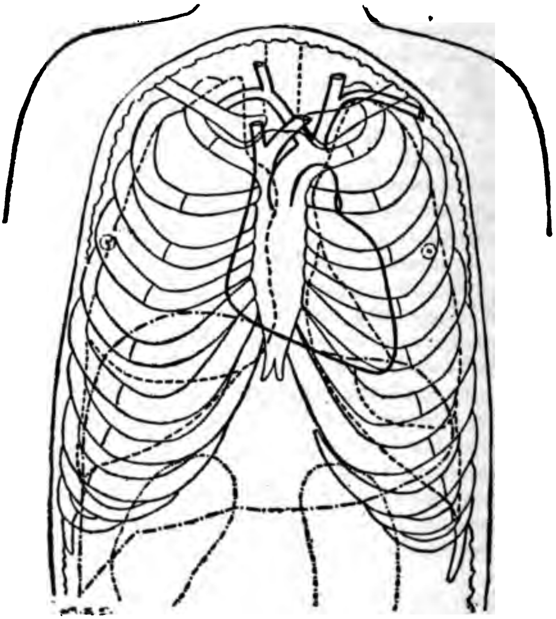

Fig. 143.—Position of viscera in child of five. (*After Symington*, "Topographical Anatomy of the Child," Plate XII.)

3. **Circulatory system.**—Note that the **apex beat** of the child is normally rather higher than in *the adult.* It is usually in the fourth space just outside the mammary line (Fig. 143). It should also be

observed that alterations in the general contour of the **præcordia** are much more frequent results of cardiac disease in children than in adults. As regards **auscultation,** it should be remembered that the pulmonary second sound in the young child is normally rather louder than the aortic. The pulmonary second is accentuated if it be permanently louder than the first. The aortic second is accentuated if it be as loud as the pulmonary.

Remember also that hæmic bruits are very rare in babies, while congenital bruits are relatively very frequent. We have already mentioned that the cardiac rhythm in the child is not unfrequently irregular even in health.

4. The **blood.**—It is sometimes difficult to get a large enough drop of blood from the ear of a child. In that case, a piece of woollen thread should be twisted round the thumb—not too tightly—and the latter punctured with a triangular needle at the root of the nail. In very young children nucleated red blood corpuscles are normally present in the blood in small numbers.* The leucocytes are more numerous in the child than in the adult; 12,000 per cmm. is not uncommon. The uni-nucleated leucocytes are relatively more abundant than in the adult. In newly-born babies the percentage of hæmoglobin is often very high.

5. **Respiratory system.**—A child uses its diaphragm much more than its intercostals in breathing. Hence the movements are chiefly abdominal, and there is little real chest expansion. Any indrawing of the lower interspaces on inspiration should always be looked for. It occurs whenever there is obstruction to the entrance of air (*e.g.* diphtheria), but may also be present where there is collapse of the lower parts of the lungs, and also in

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* This is only true during the first few days of life.

pneumonia. In "extra-auscultation" one should be on the outlook for any stridor, and for the existence of a short, grunting expiration. The latter is a frequent sign of severe respiratory disease. In the adult the normal cycle of respiration is, of course, inspiration, expiration, pause. In the child this is often reversed, so that one gets first a short expiration, succeeded by a longer inspiration, and then by a pause. This reversal is specially frequent in respiratory disease or embarrassment. The respiratory pauses are often very prolonged in the child, so that one has to wait a long time if one is ausculting before the next inspiration is heard. The normal breath sound in the child is, after the age of six months or so, **puerile** in type. **Vocal resonance** is often difficult to estimate. In babies one may make use of the cry as a producer of vocal resonance; in older children one may ask them their name, get them to count, etc. It should be remembered as a general rule that if the breath sounds are distinctly harsher on one side than the other, then the harsh side is probably the normal. Children's chests conduct sound very readily. Hence abnormal sounds, especially crepitations, are very apt to be heard on both sides, although they are really only being produced on one. The great frequency of collapse of part of the lung should be borne in mind in diagnosing pulmonary disease in infancy. In percussing the lungs in children one must, as already mentioned, use a very light stroke. One should also take care only to percuss when the chest has been filled by an inspiration, otherwise one may be led into thinking that there is dulness present.

It should also be remembered that the chest-wall of a young child is so elastic that one can often obtain the "cracked-pot" sound on heavy percussion, even although the lung be perfectly healthy. This is especially apt to occur if the child is crying.

6. **Urinary system.**—It is difficult to collect the total quantity of urine passed by a child per diem. A rough table of the average quantity for each child will be found at page 297. Sugar is very rarely present in the urine of children, but albumin is often met with, even in healthy babies.

7. **Nervous system.**—Motor **paralysis** is to be made out by watching whether the child ever moves the suspected limb. One cannot estimate the paralysis as one does in adults by means of passive resistance. Remember that inability to walk is not necessarily a sign of paralysis of the legs. One must note whether the legs are moved when the child is sitting or lying. Thus a rickety child may not be able to walk, but moves his legs freely if one tickles the soles. A child with infantile paralysis of the legs cannot move the limbs in any circumstances.

The **knee jerks** in little children are best elicited by placing the child's foot on one's hand as a stirrup, and then gently percussing the tendon. The latter lies rather to the outer side in the child, and is comparatively narrow, so that one may easily miss it.

The **superficial reflexes** are usually more brisk in healthy children than in adults. The exact localisation of **sensory paralysis** is extremely difficult in children, but sensory lesions occur but rarely in infancy.

In examining the **eyes** with the ophthalmoscope, the direct method is that which it is best to employ. The child may be examined while lying on its back, a lamp being held alongside the head, but at a somewhat lower level. It may be necessary to hold open the lids, but as far as possible avoid touching the child at all. One must often be satisfied with mere fleeting glimpses of the disc.

In testing light perception in little children, it is best to hold a candle in front of the eyes, and see if they attempt to follow its movements. One may also threaten the cornea by suddenly bringing the finger near it, and observing whether the child winks before the eye is touched.

In examining the **ears,** one must remember the shortness of the auditory meatus in the child, and the great obliquity of the drum membrane. The magnification of the view by means of an ophthalmoscopic lens is a useful aid in these cases.

It is often difficult to gauge the **intellectual capacity** of a young child. Early signs of idiocy are—inability to support the head, which often rolls about helplessly; causeless screaming; inability to notice things; and backwardness in grasping.

In older children we can inquire as to progress at school, etc., or ask the patient questions. Get him to count, multiply, and so on. The position of the child in the school is a rough guide to the development of the intelligence. A normal child remains in the infant school until he is seven years of age, after which he enters the standards. The average age of the children in each standard is as follows:—

Standard	I.	...	...	7- 8 years.
,,	II.	...	...	8- 9 ,,
,,	III.	...	...	9-10 ,,
,,	IV.	...	...	10-11 ,,
,,	V.	...	...	11-12 ,,
,,	VI.	...	...	12-13 ,,
,,	VII.	...	...	13-14 ,,

A normal child should have begun to walk a little by the age of eighteen months. Talking begins at a variable time after this. The distinction drawn by West between children that are idiotic and those that are merely backward may also be of help in the investigation; a backward child would be normal for a younger age; an idiot would be abnormal at any age.

CHAPTER XIII.

THE EXAMINATION OF PATHOLOGICAL FLUIDS.

IN this chapter we propose to deal with the method of examining fluids, which may be obtained from one of the body cavities or from abnormal growths, in order to obtain information which may be of help in diagnosis.

The fluid is obtained by means of "**exploration.**" An ordinary hypodermic needle may be employed, but special "exploring" needles—which are really merely large and strong hypodermics—are also sold. The needle should be of such calibre that it is capable of sucking up oil. If it is able to do that, it will be able to suck up any fluid likely to be met with in exploring. Before being used, the needle should be sterilised. This is best done by slipping it into a test tube, covering it with water or weak carbonate of soda solution, and boiling for three minutes. It should then be placed in boracic lotion. The use of strong carbolic lotion for sterilising exploring needles is to be avoided, as it produces a coagulation of albuminous fluids, which may block the needle or cause the fluid to seem opalescent. The patient's skin should be cleansed with some 1 in 20 carbolic acid at the spot selected for puncture. As a rule, it is not necessary to employ any local anæsthetic. In very nervous patients a small spot of skin may be frozen by means of the ethyl chloride spray. It should be remembered, however, that the local reaction after freezing causes really more pain than the original puncture. The needle should be held short, with the forefinger of the operator resting on it near the *point. It should* be introduced rapidly and steadily, but without any "stab." When the needle has been fully entered, the piston is withdrawn. Should no fluid be obtained, the needle is drawn slowly outwards, whilst a negative pressure is maintained in the syringe. It may then be found that fluid is obtained nearer the surface.

WHERE TO PUNCTURE.

In the case of the **pleural cavity,** the puncture is best made in the 9th or 10th space midway between the posterior axillary and scapular lines, this being the point at which fluid which is lying free in the cavity is most likely to be obtained. In cases of localised dulness, one must be guided, of course, by circumstances. Usually, however, one selects that point where the dulness is most absolute and the breath sound faintest.

Puncture of the **peritoneal cavity** may be performed either in the middle line through the linea alba, or laterally, about a point on a line with, but rather above, the anterior superior spine. The former position ensures that no large blood-vessel will be injured; but by lateral puncture one is more certain of entering fluid, especially if the patient be turned over somewhat on to the side of operation. Before puncturing in the middle line be sure the bladder is empty, and never insert a needle at any point unless it yields a dull note on moderately heavy percussion.

In puncturing the **pericardium** one should select a spot in the third, fourth or fifth interspace, at a distance of ½ in. to 1 in. from the left margin of the sternum, the exact spot depending on the degree of distension of the sac.

Lumbar puncture is a method now not unfrequently resorted to for ascertaining the character of the effusion in cases of increased exudation into the subdural space. It is most usually required in children. An antitoxin needle is best for the purpose. The patient, preferably anæsthetised, should be on his right side, slightly bent forward, and lying over so that the spine is towards the operator. Trace the last rib back to the twelfth dorsal spine, and count down from this to the third lumbar. Put the left thumb on the third interspinous space, enter the needle ⅓ in. to the right of it, and pass slightly inwards and upwards for a depth of ⅔ in. to 1 in., depending on the age of the patient. A syringe is not essential, but forms a useful handle to the needle. The fluid usually escapes in drops; a continuous flow indicates increased pressure. Lateral movement of the needle should be avoided, as it may produce bleeding. The fluid should be collected in a sterilised test tube. Its characters and their significance will be referred to later.

In the exploration of **cysts,** etc., one must be guided by local circumstances, the rule being to select for puncture that part of the tumour which is nearest the surface, and where one is not likely to injure important structures.

EXAMINATION OF THE FLUID.

The fluid having been obtained, it should be transferred to a conical glass and allowed to settle.

Note first its **physical characters.** The chief of these are the colour, consistence, specific gravity, odour, and the appearance of the deposit (if any).

As regards the **colour** of the fluid, one of the most important points to note is whether it is bloodstained or not. It must be borne in mind, however, that a small amount of blood is apt to get into the fluid in the process of exploring. Observe, also, whether the fluid is transparent, opaque, or opalescent.

Opacity is usually due to the presence of cellular elements; opalescence to fatty particles or large numbers of micro-organisms.*

Opacity or opalescence due to fat may be removed by adding to the fluid some caustic potash solution and then shaking up with ether. The fat is dissolved out, and if the ether is sprinkled on to blotting paper leaves a stain. Fluid which is opaque from the presence of much fat is usually spoken of as "chylous."

Pathological fluids are usually of a more or less watery **consistence.** Viscidity usually indicates the presence of mucin. It should be carefully noted whether or not the consistence of the fluid alters on standing. Many pathological fluids clot after standing for some time. The clot consists of fibrin.

The **specific gravity** is taken with a urinometer; the same precautions being used as in the case of urine (p. 303).

Most fluids are devoid of **odour**; sometimes, however, they are extremely fetid.

The amount and colour of the **deposit** should be noted. If red, it probably consists of red blood corpuscles; if white, it may be made up of leucocytes, cancer cells, etc.

For **chemical investigation** the fluid should first be filtered. In the examination of the filtrate the following points must be attended to :—

1. The **reaction.**—This is almost invariably alkaline. Sometimes it is neutral.

2. The presence of **serum albumin** and **serum globulin.**—This is ascertained by means of the same tests as have already been described for the urine (p. 328). If these proteids are present in large amount, the fluid is coagulated on boiling, even although the reaction is alkaline. If proteids are scanty, the fluid

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* Opalescence may apparently be due also to the presence of *peculiar forms* of proteid and of lecithin.

should first be rendered slightly acid by means of dilute acetic acid.

As in the case of the urine, nothing is gained by testing for albumin and globulin separately. **Albumoses and peptone** are almost never found in the fluids under consideration.

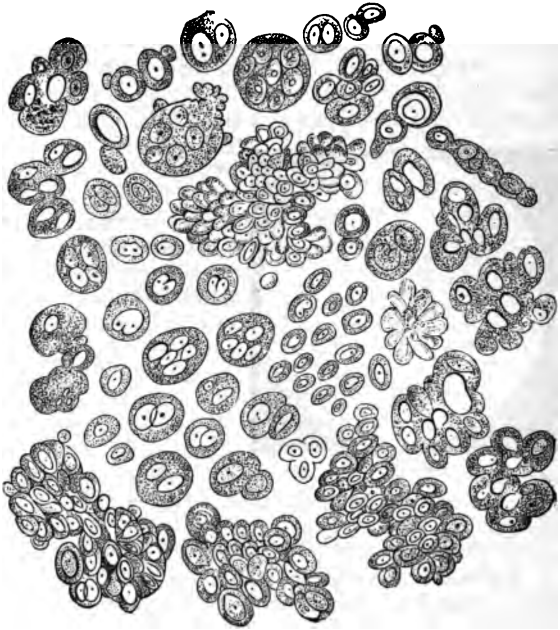

Fig. 144.—Foulis' cells.

The quantitative estimation of albumin and globulin cannot be accurately carried out in ordinary clinical work. Approximate results may be obtained by the use of Esbach's tube. The fluid must first be very freely diluted, so as to bring the specific gravity down to 1008, and should then be rendered acid by means of acetic acid (*see* p. 331).

3. The presence of **mucin** or **nucleo-albumin** is proved by the appearance of a precipitate on the addition of acetic acid. The method of distinguishing between the two has been already indicated (p. 335).

4. **Sugar** should be tested for by rendering the fluid slightly acid, boiling, and filtering. The filtrate is then evaporated down to a small bulk, and the tests for glucose described at pp. 341–346 applied.

5. **Urea** is not often present, except in traces, in ordinary pathological fluids. In fluids derived from the urinary organs it may be more abundant, and should be tested for by removing all proteids by heat, evaporating the filtrate to a small bulk, and then testing for urea as described on p. 315.

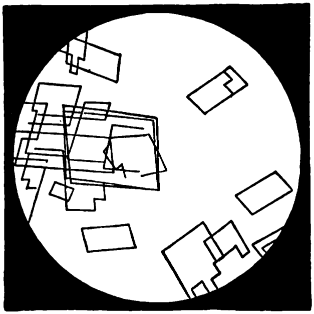

Fig. 145.—Cholesterin crystals.

MICROSCOPIC EXAMINATION OF THE SEDIMENT.

Some of the deposit is taken up with a pipette, and a drop of it placed on a slide, covered and examined. If desired, films of it may be made in the same way as in the case of blood (p. 220). This succeeds fairly well if the deposit consists of cancer cells.

One may recognise under the microscope (1) elements derived from the **blood**—altered red and *white corpuscles*. The recognition of altered white *corpuscles or* pus cells is facilitated by mixing with a drop of the deposit a small quantity of a 1 per cent solution of acetic acid to which a little methyl green has been added. The nuclei are then more easily recognised. (2) **Epithelial cells** of various sorts. The recognition of cancer cells is of special importance. The addition of a little picrocarmine or magenta facilitates the process. Do not mistake ordinary endothelial cells for them. Cancer cells should be large, numerous, and show grouping here and there. The cells met with in fluids derived from malignant ovarian cysts, or malignant peritonitis following such cysts, are known as Foulis' cells (Fig. 144). They are large cells containing one or more nuclei about the size of a red blood corpuscle. They may either have a smooth outline or may show little buds or projections indicating rapid proliferation. Very similar cells are met with in effusions due to malignant disease of the pleura. In fluids derived from hydatid cysts,

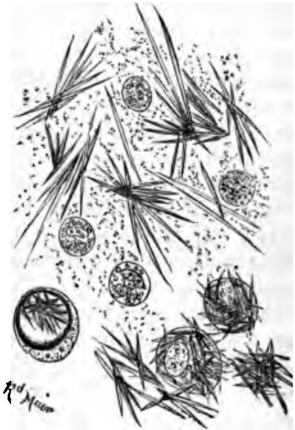

Fig. 146.—Fatty needles and fatty crystals in degenerated cells.

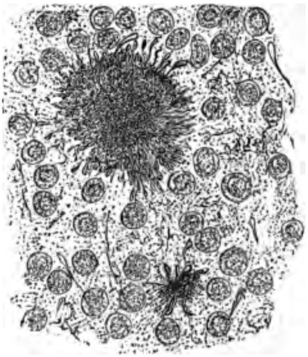

Fig. 147.—Colony of actinomyces in pus, from a case of actinomycosis of the cæcum. × 300.

scolices and **hooklets** may be found (p. 103) (3) **Crystals**—*e.g.* of cholesterin (Fig. 145), or of fatty acids (Fig. 146), fragments of muscular tissue, etc.—are sometimes met with. (4) The pus from cases of **actinomycosis** contains small seedlike nodules. If one of these is bruised between a slide and cover glass, and examined with the high power, it will be seen to consist of a central mass of detritus, radiating out from which are a number of club-shaped highly refractile bodies (Fig. 147). If there is any difficulty in identifying the fungus, cover-slip preparations should be made, and stained by Gram's method (Appendix, 20). For other staining methods, *see* p. 590. (5) The detection of the **Amœba dysenteriæ** in the pus of abscesses, especially in the liver, is often of great diagnostic value. The organism is described at p. 106. If one fails to find it in pus removed by exploration or in that obtained when the abscess is first opened, one must not jump to the conclusion that it is not present in the abscess. It frequently does not appear in the discharge until three or four days after the abscess has been opened. This is probably to be explained by the fact that the habitat of the organism is in the wall of the abscess (Manson).

GENERAL CHARACTERS OF THE PRINCIPAL FLUIDS.

1. Inflammatory and dropsical effusions.

Inflammatory effusions are often spoken of as exudates; dropsical effusions as transudates. They present the same general appearances, being clear fluids of a yellowish green colour, and containing much albumin and globulin. It is very difficult to tell a dropsical from an inflammatory fluid by chemical or other examination. It would appear that the amount of proteids in an effusion depends much more upon site than upon cause. Pleural fluids contain the highest percentage of proteids, peritoneal fluids rather less, and subcutaneous fluids very little. The fluid in cardiac dropsy is more highly albuminous than in dropsy of renal origin. From a diagnostic point of view all one can say is that a fluid with a specific gravity of more than 1018, which contains more than 4 per cent. of albumin, is almost certainly inflammatory; while one with a specific gravity of less than 1015, and an albumin percentage of less than 2½, is certainly dropsical. Between these limits one must be in doubt. Nor is the occurrence of coagulation in the fluid after tapping of much help. If the coagulation be very rapid and complete, the fluid is probably inflammatory, but considerable coagula may form even in purely dropsical fluids after standing for some time.

Variations in the specific gravity, etc., of fluids obtained by repeated tapping in the same case are of no prognostic value. A marked rise in specific gravity and amount of albumin may, however, indicate the supervention of inflammation.

2. **Fluid obtained by lumbar puncture.**—Ordinary cerebro-spinal fluid is perfectly clear and colourless, resembling distilled water. It contains very little albumin ($\frac{1}{50}$ per cent.), and if evaporated to a small bulk and tested will be found to reduce Fehling's solution, owing, probably, to the presence of pyrocatechin. This is the kind of fluid which is found in cases of cerebral tumour. In cases of meningitis the fluid is not clear, but turbid from the presence of cellular elements. The turbidity may be so slight that it is only noticed after shaking the fluid and holding it up to the light. It contains quite an appreciable amount of albumin, varying from ½ per cent. in chronic cases up to 1 or 2 per cent. in those which are acute; and a small clot of

	HYDATIDS.	CEREBRO-SPINAL FLUID.	HYDRO-NEPHROSIS.	DISTENDED GALL BLADDER.	OVARIAN CYSTS.	PAROVARIAN CYSTS.	PANCREATIC CYSTS.	AMNIOTIC FLUID.
Colour ...	Colourless or slighty opalescent	Colourless	Colourless or yellowish	Colourless or bile-stained	Varies — brown, green, yellow, etc.	Colourless	Colourless or yellowish and turbid	Greenish yellow
Consistence	Watery	Watery	Watery	Slightly viscid	Viscous	Watery	Watery	Watery
Specific gravity ...	1006–10	1007–8	1008–20	Low	1002–50	Low	Low but variable	1006–11
Coagulable proteids	Very scanty	Very scanty	Vary — may be fairly abundant	Usually sconty	Vary	Scanty	Variable	Scanty
Special characters	Presence of scolices or hooklets	Contains a cupric oxide reducing substance	*May* contain urea or uric acid	May contain bile. Mucin usually present	Presence of pseudomucin (gives a white precipitate with alcohol after other proteids have been removed by boiling)	—	Contains cholesterin, and (if recent) will digest egg-albumin in alkaline medium, and may convert starch	Heavy animal odour. Contains some urea (at least in later months)

fibrin usually appears in it after standing for some time. It does not usually contain any reducing substances. The bacteriological examination of the fluid is of special importance, and should be carried out as in Chapter XIV.

3. The chief characters of the other fluids likely to be met with on exploration are contained in the foregoing table.

CHAPTER XIV.

CLINICAL BACTERIOLOGY.

IN order to make a complete bacteriological examination, three methods of observation are necessary. These are microscopic study of the organisms, their cultivation on suitable media, and the effects produced by their inoculation into various animals. To obtain material, and to carry out the subsequent examination, the following instruments, all of which must be thoroughly sterilised immediately before use, are required :—

1. **Platinum needles,** about 3 in. in length and sufficiently stout not to bend very readily, fused into glass handles. Some of these should have their ends curved into a small loop, others ought to have the last half centimetre bent at right angles to the rest of the wire, whilst a few should remain straight and have their free extremity somewhat flattened like a narrow spatula.

2. **Small sterile pipettes.**—For clinical use, ordinary vaccination tubes, carefully sterilised and then sealed off, are very serviceable. A few larger ones are also needed.

3. **Swabs** for collecting particles from false membranes. To make these, a piece of stiff copper wire, about 1½ millimetres in diameter (No. 17 standard wire gauge), should be employed. A portion, 7 in. long, should have a flat loop made at one end, round which a small piece of absorbent cotton wool must be firmly twisted. Wool must not be used that has been rendered antiseptic with mercuric chloride or other disinfectant. A test tube about an inch shorter than the wire is then

selected, and its mouth plugged with a stopper of cotton wool, through which the wire passes. Thereafter the tube with its contained swab is carefully sterilised by heat. Several such swabs should be taken to the patient, and, after having been used to collect the material which requires examination, can be replaced in their test tubes and brought back to the laboratory without fear of contamination.

4. A **hypodermic syringe** which can be sterilised by boiling. One which the writers have found convenient is designed by Strauss, and sold by Collin of Paris. The plunger is made of elder pith, and by a simple adjustment can readily be fitted to the piston rod, and the other joints are also rendered secure by discs of the same substance. Boiling improves rather than injures the pith fittings, and when one has worn out, a new disc can easily be cut with a penknife from a piece of fresh pith.

In order to sterilise the swabs, test-tubes, and other apparatus, a small dry heat oven is required; the workroom should also contain a centrifuge, test-tubes, beakers, flasks, funnels, filter papers, a Bunsen burner, capsules, watch-glasses, forceps, slides and cover slips, stains and reagents, and a good microscope, magnifying at least 500 or 600 diameters. If cultures are to be made, an incubator and a number of other accessories are necessary in addition to those already enumerated.

The **capsules** should be of porcelain, nickel, or platinum, and should be employed, wherever heat has to be applied to staining fluids, in place of watch glasses, which crack readily. Metal capsules should be scrupulously washed, and then sterilised by heating to redness in a flame. Porcelain ones should also be sterilised by heat, and must not be used after they become cracked.

Slides and cover glasses.—The latter must be thin, preferably No. 1, and both should be cleansed with Van Ermengen's solution. Thereafter they may be kept in a covered vessel under alcohol until they are required. (*See* Appendix, 26.)

The most generally useful **forceps** for holding cover glasses are those known as Cornet's. In these the spring is so arranged that the cover glass is firmly held until released by pressure, and the blades are constructed so that the cover slip is in a horizontal position when the forceps are laid down. The two blades of this forceps should be dissimilar, one side having an aperture punched in the spring. The film side of the cover glass should invariably correspond with the aperture; this obviates all risk of error in preparing and mounting the specimen. In selecting a pair of Cornet's forceps one should reject any which when laid on the table fail to hold the cover slip horizontally, and also those the ends of whose blades meet at a somewhat acute angle, as they are less secure in their grip, and are apt by capillary action to draw off the stain from the film.

The **stains** which are most employed belong to the basic aniline dyes, and are either simple aqueous and alcoholic solutions, or contain a mordant, such as an alkali, carbolic acid, or aniline oil, which makes the bacteria take up the stain better and retain it more firmly. Most of these dyes are so used that the preparation is first overstained and then the excess washed out by suitable reagents, when, owing to the greater tenacity with which bacteria cling to the colouring matter, they remain clearly visible after all else has become partially decolorised. A few stains, however, *e.g.* Bismarck brown, are employed differently, as their selective action is so marked from the very outset, and their tendency to overstain *is so slight*, that there is no need of securing differ*entiation* by the more tedious method of overstaining and then washing out. Such stains generally act with fair rapidity.

For most purposes the following stains suffice :—

1.	Fuchsin	saturated alcoholic solution.
2.	Gentian violet	saturated alcoholic solution.
3.	Methylene blue	saturated watery solution.
4.	Bismarck brown	aqueous solution.
5.	Löffler's stain	see Appendix, 20–23.
6.	Ziehl-Neelsen's stain [for tubercle]	
7.	Carbolthionine	
8.	Gram's reagents	

The stains must not be kept too long, must be frequently filtered, and should be examined from time to time for bacteria, which occasionally invade them—especially when the solutions are aqueous—and unless detected may lead to serious mistakes.

For mounting the specimens one may employ Farrant's medium if the slide is not to be permanently preserved, otherwise Canada balsam in xylol should be used. It is very important to remember that some stains make bacteria look larger than others do, and also that their apparent size is less when they are mounted in balsam than when the examination is made with a drop of water or with Farrant's medium. When cultures are desired, tubes containing nutrient gelatin, blood serum, agar-agar, and glycerine agar, and flasks of bouillon must be provided. These can occasionally be obtained from dealers, but are better prepared in a well-equipped laboratory. The details of preparation are outside the scope of this text-book.

The **method of examination** which is most readily available in clinical work is the preparation of **films** on cover slips. The technique is as follows:—A cover slip, which must be thin enough to admit of *the film being* examined through it by an oil immersion lens, and which is thoroughly clean, is taken, and a very small drop of the fluid under examination is spread over it in a thin layer by a sterilised platinum needle. If the material is too solid, as may be the case when cheesy particles are being examined, a drop of distilled water must be added and intimately mixed with the mass before the cover slip is smeared with it. The excess of fluid is then allowed to evaporate by holding the preparation, film upwards, some height above a flame, and the *dry* film is "fixed" by passing it thrice through a Bunsen or spirit flame, film upwards, at such a rate that each transit occupies something less than a second.

Thereafter, in order to make the background stain less deeply, it is immersed for one minute in a 1 per cent. aqueous solution of acetic acid. It is then quickly dried, and fixed in the forceps, film upwards. A test tube is taken and half filled with distilled water, and the selected stain, which should have been recently filtered, is added until the fluid is just transparent. Take up a few drops of the dilute stain with a pipette and apply it to the film. To avoid a ring of deposit on the cover slip, sufficient stain must be applied to cover its entire surface. After the lapse of a couple of minutes wash the slip with water, dry it thoroughly, and mount in xylol balsam.*

The specimen must be examined with a high-power objective. In most instances an oil immersion lens of $\frac{1}{12}$ inch focus should be used, although in many cases a lower power, such as a Zeiss D objective, may be sufficiently strong for clinical purposes. The microscope should be provided with an Abbé condenser the diaphragm of which must be used fully opened, and the plane mirror employed to reflect the light. The inner tube of the microscope should be drawn out to the length for which the objective is constructed. Continental objectives mostly work to best advantage with a tube length of 160 mm. English objectives require a tube length of 10 inches.

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* *Some* stains, *e.g.* Löffler's methylene blue, take much *longer; for* details, *see* Appendix, 22.

In using the oil immersion lens, a drop of prepared cedar oil is placed on the cover glass, and the objective lowered by the hand or coarse adjustment until it touches the surface of the oil; the focussing is then carefully performed with the fine adjustment. As the lens is delicate and the working distance small, great care must be taken not to bring the lens and cover glass into contact; and when the observation is completed the cedar oil must be gently wiped from the surface of the objective with a piece of old silk.

In examining a hanging drop preparation, such as is used in testing for typhoid, care must be taken not to break the unsupported cover glass in focussing with the oil-immersion lens. Curtis* advises the following procedure in such cases. Find the edge of the drop with a low power objective, and observe that beyond this there is an area of minute points of water condensed on the under surface of the cover slip. This dew should be brought to the centre of the field, the low power lens replaced by the oil immersion, and the latter directed upon the dew, which is much more easily focussed than are the contents of the hanging drop itself. The slide is then moved, *without altering the focus*, until the drop occupies the field of vision, and its contents both near the centre and towards the periphery can be examined without any risk of breaking the preparation. Once the focus of the upper surface of the drop has thus been defined, careful use of the fine adjustment will readily bring any other plane of the preparation into sight.

When a tube requires to be inoculated the following procedure should be adopted :—

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* *Curtis*, "Essentials of Practical Bacteriology," p. 31.

The tube containing the pathological fluid is held between the thumb and first finger of the left hand, so that the mouth with its stopper of cotton wool is on the palmar side. The tube to be inoculated is similarly held between the first and second fingers of the same hand, with the surface of the nutrient medium upwards. Both tubes should be held as horizontally as possible short of wetting the stoppers with the contents. A platinum needle is then taken in the right hand, thoroughly sterilised by heating to bright redness, and allowed to cool again. The stopper of the specimen tube is then withdrawn by the right hand and placed between the fourth and fifth fingers of the left hand, and finally the stopper of the nutrient tube is withdrawn and retained between the fingers of the right hand, care being taken that the stoppers are only held by their external ends. The needle is then dipped into the specimen, and the smallest trace of it is withdrawn and transferred to the surface of the nutrient medium, along which a streak is drawn. The tubes are at once re-stoppered, the needle again sterilised, and the culture placed in an incubator as soon as convenient.

When micro-organisms are very abundant in the specimen, several tubes should be successively inoculated without recharging the needle. In this way, though the growth is excessive in the first, in the third or fourth the colonies are more scattered, and pure cultures may be separated out.

A cheap metal pen-rack is a convenient support on which to lay the needle after it has been sterilised, when a number of tubes are being dealt with.

SPECIAL METHODS.

1. **Sputum.**—The sputa which are of the *greatest* importance bacteriologically are those of pneumonia and of phthisis. In pneumonia the diplococci are most readily found when the disease is at its height. Select a rust-coloured portion of sputum, spread it in as thin a film as possible, dry, fix, and stain with the fuchsin solution employed for tubercle bacilli as recommended by Ziehl. Decolorise by means of warm water. If successful, the cocci will be darkly stained, and the surrounding capsule will appear in a fainter shade of red.

In tubercular sputum much of the success depends on the choice of a suitable specimen. The sample should be chosen from the interior of one of the mucopurulent masses that are seen underneath the serous fluid portion. If the mass is so tenacious that a suitable piece will not adhere to the needle, spread the sputum in a shallow glass vessel and either pick out the part required with sterilised forceps or plunge a hot needle into the mass, when a portion will adhere to it; care must be taken not to use the central charred mass. When the sputum comes from a phthisical cavity it may contain almost pure cultures of tubercle bacilli in its interior, though the outer surface of the mass is naturally contaminated during its passage through the mouth. To prevent confusion from spread of contamination, the sputum ought always to be examined as soon as possible after it has been expectorated. (FRONTISPIECE, *b.*)

In cases of hæmoptysis the best specimens in which to seek for bacilli are often the small dark red clots that are coughed up a day or two after the attack has subsided.

In acute miliary tuberculosis without typical tubercular sputum, it may be almost impossible to demonstrate the presence of the bacilli.

In difficult cases the sputum should be heated with caustic soda and water till a homogeneous fluid is produced. This is set aside for thirty-six hours in a conical test glass and the sediment examined, or the sedimentation may be more rapidly effected by a centrifuge.

When a suitable specimen has been obtained, it should be spread very thinly and evenly on a cover glass, and allowed to dry in the air at the temperature of the room. *When it is quite dry*, it is taken in a forceps and passed several times rapidly through a smokeless flame, as has been already described, to coagulate the albuminous materials, and so fix the film to the glass. It should then be placed film downwards on the top of a little carbol-fuchsin stain contained in a capsule, and the fluid heated till steam rises. Generally, it is well to repeat the heating three or four times. Then wash in water and decolorise in a 20 per cent. solution of sulphuric acid until all the elements except the bacilli have parted with the stain. As a rule, this is accomplished in from one to three minutes. Wash once more in water, dry, and mount either in a drop of Farrant's medium if the specimen is not to be kept for any length of time, or in Canada balsam dissolved in xylol if a more permanent preparation is required.

The specimen must be examined with a high power and good illumination; by preference with an oil immersion lens and Abbé condenser—though for clinical work a lower power is often sufficient.

Many workers prefer to counterstain with a watery solution of methylene blue for one minute. This has the advantage of revealing the other elements present in the sputum, and the disadvantage of making the detection of the tubercle bacilli rather more difficult.

2. **Blood.**—This may be readily examined by careful sterilisation and subsequent pricking of the *finger-tip*, so as to make a drop of blood exude *with which to* prepare films on cover glasses. Since

it is often very difficult to obtain complete disinfection of the skin, an alternative method may be adopted, and blood aspirated from a vein in the arm by means of a syringe. In this case the syringe, which should have a capacity of at least 5 cc., must be rendered thoroughly aseptic by boiling before use. As steel needles are rapidly rusted by exposure to the air after being boiled, they may, on removal from the water, be plunged into a tube of absolute alcohol and kept there until required, or, instead of boiling water, hot oil may be employed to sterilise them. The median basilic vein is usually selected as suitable in size and position, and after the surrounding skin has been scrupulously cleansed it is rendered prominent by applying a proximal bandage to the arm. The needle is quickly but steadily introduced, care being taken that its point remains in the lumen of the vessel, and the required amount of blood is abstracted. This should immediately be transferred to a large tube containing from 20 to 30 cc. of glycerine agar, prepared with a sloped surface; the tube can then be taken to the laboratory for further examination. This method should always be adopted when the recognition of bacteria in the blood is of importance, not only because in a certain number of cases it is difficult to avoid contamination when the blood is derived from a finger-prick, but also—which is much more important—because in many instances the number of bacteria in the circulation is comparatively small, and unless a considerable quantity of blood is employed the sample may be quite free from the organisms and a negative result may be erroneously recorded Puncture of the spleen has been advocated on the Continent, but it may lead to unpleasant symptoms.*

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* Similarly, some observers have punctured the lungs, liver, and other organs to secure uncontaminated samples of the bacteria which they contained.

When the film of blood has been made on the cover glass, it must be well dried in warm air before it is passed through the flame, and thereafter should remain for some time, best for three or four hours, in an oven at a temperature of 120° C. Where it is important to retain the morphological characters of the blood as well as to demonstrate the presence of bacteria, the moist film must be fixed by being floated face downwards on a strong solution of corrosive sublimate in normal saline fluid; or some of the special fixation methods described in Chapter V. may be employed. Before applying the stain a preliminary immersion in dilute acetic acid assists both in discharging the colour of the red blood corpuscles and in accentuating the different affinities of the bacteria and blood elements for the dye; it must, however, be carefully washed off, and the last traces of it neutralised by ammonia vapour before staining is proceeded with.

The stains which prove most useful are Gram's, Löffler's, or alcoholic fuchsin. When the stain is blue, watery eosin may be used as a counterstain to bring out the red blood corpuscles.

3. **Urine** may be examined, after careful disinfection of the meatus, either by drawing off a sample from the bladder with a sterile catheter, or by making the patient pass water, and, after the first portion of the urine has cleansed the urethra, collecting the remainder. The centrifuge should be invariably used to secure a deposit without delay, and the latter then examined.

One of the most important bacteria that may be found in the urine is the tubercle bacillus. The pus which is separated from the suspected urine is spread not too thinly on a cover glass, which is then manipulated in the manner already described for tubercular sputum. In cases where the sediment contains small purulent-looking lumps, these should be selected to smear on the cover glass. If the urine is loaded with urates they may be readily dispelled by adding warm water to the sediment, or by washing the film with warm water before staining.

The bacilli when found frequently occur in clumps, whilst it may be necessary to examine six or more preparations before a clump is discovered.

One of the chief sources of fallacy in examining for tubercle bacilli results from the presence of the smegma bacillus, which has very similar morphological characteristics, and especially resists the decolorising action of sulphuric and nitric acids. To distinguish them the cover slip should be immersed for ten minutes in a solution of hot caustic soda to which 5 per cent. of alcohol has been added. The specimen is thereafter washed with water and absolute alcohol. Tubercle bacilli thus treated still retain the stain when exposed to the action of mineral acids, whilst the smegma bacillus is decolorised.

In cases of cystitis the most commonly found organisms are the bacterium coli, the gonococcus, and the tubercle bacillus. In some instances, especially in women or in persons on whom catheters have been passed, the ordinary staphylococci of suppuration are also present in large numbers.*

In urethritis resulting from gonorrhœal infection the gonococcus is present, but is often associated with numerous other diplococci, which are not very readily distinguished from it. Since it is of importance medically as the cause of certain affections which resemble rheumatism, it is necessary to be able to recognise it. Films may be made in the ordinary way, stained lightly with watery solution of methylene blue, again washed and examined in water, or dried and permanently mounted. The organism will be described subsequently.

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* The name "Bacteruria" has been given to conditions where the urine, on being examined immediately after it is passed, is found to contain bacteria. Many writers, however, limit the name to cases where the bacteria present are not tubercle bacilli or the ordinary micrococci of suppuration. For full information, *see* Barlow, "Ueber Bacterurie," Deut. Archiv. *f. klin. Med.*, Bd. 59, s. 347.

4. **Pus and other fluid exudations** are best obtained from the cavity in which they lie by aspiration with a sterile hypodermic syringe, after thorough disinfection of the skin where the needle is to be inserted. Amongst the most important of such exudations are those into the pleural cavities. These may either be serofibrinous or purulent. In nearly three-fourths of the cases of empyema which have been examined, the organisms that have been found are either pneumococci, staphylococci, or streptococci, and in the case of adults the last constitute fully half of the total. In children the pneumococcus preponderates. As regards prognosis, the presence of streptococci gives much the gravest outlook, and this agrees with the relatively favourable issue of the disease in children. There can be no doubt that a very large proportion of pleurises and empyemata are due to tubercular infection. It is extremely difficult to demonstrate the bacilli either by culture methods or in cover slip preparations, but inoculation experiments have frequently been successful. Cases of empyema may be regarded as very probably tuberculous if agar tubes inoculated with the pus remain sterile.

5. Specimens from **false membranes** are best obtained by removing a small portion with a swab or forceps. Care must be taken not to touch any other part of the patient's mouth with the swab. In cases of suspected diphtheria it often happens that so many organisms are present on the surface of the membrane that it is almost impossible to identify

the bacillus of diphtheria amongst the other bacteria which are associated with it. Under these conditions the piece of membrane should be washed in a flask with a little boiled water. After being agitated in this for a short time, the cocci, mucus, and other adventitious elements which have adhered to the surface become for the most part detached, and the specific organism can thereafter be obtained in a much purer condition by thrusting a platinum needle into the membrane after its removal from the flask, and inoculating several tubes successively without recharging.

6. **Fæces.**—The fæces are always rich in micro-organisms; it is therefore of great importance to lift only a very minute fragment for examination, whether on the slide or by cultures.

The following are the most important bacteria from the point of view of clinical examination:—

I.—BACILLI.

1. **Bacillus tuberculosis.**—It is rather small and slender (2 to 5 μ in length). As seen in sputum it is not unfrequently a little curved; and often two bacilli are found lying end to end, making an obtuse angle with one another. The staining may be uniform, or there may be small clear spaces of an ovoid form which are disposed at intervals. It is best stained by Ziehl's method.

Recent research has shown that a considerable number of bacilli, not usually regarded as pathogenic in man, share with the bacillus tuberculosis the property of resisting the decolorising action of acids and of alcohol. This group of bacteria are described as "acid-fast," and since they possess many of the other morphological characters of the true tubercle bacillus, and have been occasionally found in sputum from the lungs, in mucus from the respiratory passages, and not very unfrequently in urine containing pus, they may give rise to errors in diagnosis. So far as is known at present the most characteristic difference is that, even at the most favourable *temperature*, tubercle bacilli grow much more slowly on culture media than do the other members of the "acid-fast" group, many of which, unlike the tubercle bacillus, can also flourish at the ordinary temperature of the laboratory. As these bacteria are fairly numerous outside the body they may readily contaminate secretions which have been laid aside for examination, and, besides this, there can be little doubt that they have often been mistaken for the tubercle bacillus in milk and other articles of diet.

2. **Pfeiffer's bacillus** is a minute bacillus with rounded extremities; the ends stain more deeply than the centre. It is not coloured by Gram's method, but can be demonstrated by Ziehl's stain. It is found in the sputum, and occasionally in the blood of patients suffering from influenza.

3. **Bacillus diphtheriæ** (Klebs-Löffler).—This bacillus averages about 3 μ in length, the long variety considerably more, and ·6 or ·7 μ in breadth. The ends are rounded and rather stouter than the centre, and stain more deeply; frequently there is unequal staining of the protoplasm. (FRONTISPIECE *a*, *a'*.) In most instances they are very readily coloured by Gram's method.

The procedure which should be adopted in cases where a patient's throat is to be examined for the bacillus of diphtheria, is as follows. Set the patient in a good light. Depress the tongue with the handle of a spoon; then take a swab, pass it to the back of the mouth without touching the lips, gums, or tongue, and press it gently against the suspected patch on the tonsil. By rotating the swab a portion of the false membrane is entangled and brought away, and the specimen thus obtained is returned to the tube and taken to the laboratory. A cover-glass preparation may be made directly from it, but is likely to prove unsatisfactory, as many different organisms are so abundantly present that it is seldom possible to detect the Klebs-Löffler bacillus with certainty. To start cultures, a platinum needle, previously sterilised and allowed to cool, is charged from the false membrane, and four tubes are successively inoculated from the needle, which is drawn in a streak along the surface of each without recharging. By far the most satisfactory medium is blood serum. Gelatin and glycerin-agar are untrustworthy, and should never be employed for diagnostic cultures of the diphtheria bacillus.* After inoculation the tubes are placed in an oven at 37° C. If the Klebs-Löffler bacillus is present, growth will often be manifest in eighteen, and always in twenty-four hours, by which time the colonies in the third and fourth tubes will be as large as pinheads. In colour they are dull white, and appear distinctly denser in the centre when viewed by transmitted light. The colonies are circular, and spread rather rapidly. In the first and second tubes the characters are not so well seen, because the colonies are so numerous that they rapidly fuse and so lose their contours. Specimens obtained from the individual colonies must be microscopically examined, and pure cultures can be started from some of them by mixing them with a little bouillon and reinoculating serum tubes with the product. In old cultures, and sometimes also in the membrane obtained from cases of diphtheria, involution forms of the bacillus are common.

In doubtful cases the diagnosis may be established only after inoculations have been performed. Neisser has described a special method of staining the diphtheria bacillus, which he considers important when a differential diagnosis has to be made. A culture of the bacilli in question must first be prepared on Löffler's solidified blood serum, at a temperature of 34° or 35° C.—temperatures over 36° C. render the reaction untrustworthy. After an incubation period of from ten to twenty hours, a cover slip preparation is made and stained for three or four seconds in the following solution:—

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* Failing blood serum, a medium composed of agar-agar prepared with ascitic, pleuritic, or hydrocele fluid, containing 2 per cent. of a 10 per cent. solution of caustic potash, with 5 per cent. glycerin, and 1 per cent. grape sugar, as recommended by *Kanthack*, may be tried.

Methylene blue	1 part.
Alcohol	20 parts.
Distilled water	950 parts.
Glacial acetic acid	50 parts.

The film is then rinsed in water, and counterstained for three or four seconds in a filtered solution of 1 part vesuvin in 500 parts of boiling distilled water. If the organisms be true diphtheria bacilli their bodies will be stained brown throughout their whole length, but one or two, sometimes three, granules of a bright blue colour, known as Babes-Ernst bodies, are also visible. Where there are only one or two granules they occupy positions near the ends of the bacillus, the third when present is situated about the middle. The granules appear to have a greater diameter than the rest of the bacillus, and their shape is slightly oval.

Occasionally, when the membrane is peculiarly rich in adventitious bacteria, it may be necessary to place it in a flask with some boiled water and to treat it in the manner described above, before proceeding to inoculate the tubes.

When information is sought regarding other bacteria which may also be present in the specimen, it is best to break up a small fragment in bouillon and inoculate various culture media with a drop of the fluid.

In practice one finds that sometimes the membrane contains a preponderant proportion of Klebs-Löffler bacilli; in many cases, however, other *bacteria* occur in large numbers along with it, *streptococci* being frequently present, particularly in severe cases. In other instances a form of diplococcus has been observed.

In addition to the typical form of Klebs-Löffler bacillus there is a stunted form which is shorter and more irregular in outline. One cannot, however, estimate with any certainty the toxicity of a specimen from its morphological characters, nor, on the other hand, is one justified in considering that the bacillus is not a true diphtheria bacillus because it happens to be devoid of virulence, although the intensity of its toxin will obviously influence the clinical history of the case from which it is derived. It is not therefore expedient to classify such non-virulent bacteria as "pseudo-diphtheritic," since experience has shown that the virulence of diphtheria bacilli may vary exceedingly even when they have sprung from a common source.

The term "pseudo-diphtheritic bacillus" has also been applied to various bacteria whose morphology is almost identical with the true, especially the short form, but which, under the tests of culture and inoculation, are found to deviate more or less from the latter. Used in this sense the term is convenient, if not strictly accurate. As alkaline bouillon containing a trace of glucose is gradually rendered acid by the true diphtheria bacillus, the change usually occurring within two days, whilst many of the "pseudo" forms do not alter its reaction, the test may be employed within certain limits to discriminate between them.

4. **Bacillus of typhoid** (Eberth). — This organism can be obtained in the spleen during the course of the disease, and from the blood in the rose-coloured spots. It also occurs in the urine if albuminous, and in the stools. It is mobile, and by suitable methods flagella can be demonstrated. The *ends are rounded*, the length 3 or 4 μ, the breadth

about 1 μ. It does not stain by Gram's method, but is readily coloured by the ordinary aniline basic dyes.

In arriving at an early diagnosis of typhoid fever **Widal's test** seems likely to take a very prominent position. It is based on the fact that after a certain time has elapsed the blood serum of a typhoid patient acquires a power of interfering with the ordinary behaviour of the bacillus, and produces the phenomenon known as *agglutination*. It is conducted as follows:—

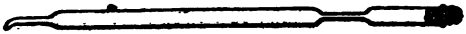

Fig. 148.—Widal's Pipette. Actual size.

(1). **Apparatus.**

(*a*) A small lancet-shaped needle to obtain blood. This must be capable of ready sterilisation.

(*b*) A sterile pipette to collect the blood and transmit it to the laboratory.

(*c*) A platinum needle ending in a loop of 1mm. diameter. (This will lift about 2 mgms. of fluid.)

(*d*) Several sterilised watch glasses, slides, and cover glasses.*

(*e*) A supply of sterile physiological salt solution.

(*f*) A tube of bouillon containing a culture of typhoid bacillus not more than twenty-four hours old. The tube must be free from clumps of bacilli, and the latter must exhibit active movements on microscopic examination. Just before the test is commenced some of the bouillon should be passed through a moistened filter paper to get rid of any stray clusters of bacilli; a bouillon which before filtration contains many clumps should, however, on no account be used if it is at all possible to obtain a fresh supply.

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* An ordinary slide may be used if care is taken not to press down the coverslip unduly; or a slide with a central depression may be preferred, and a hanging drop preparation employed.

(*g*) A microscope capable of magnifying at least 250 diameters (preferably from 300 to 400 diameters) should be used, though even with lower powers a good deal may be made out.

(2). **Method.**

(*a*) Carefully sterilise the patient's finger, and obtain several drops of blood by a prick in the thin skin near the root of the nail. The blood is drawn into the pipette, which is then sealed off and taken to the laboratory.

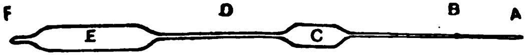

Fig. 149.—Wright's Pipette. One-half actual size.

A, open end of capillary tube; B, index; C, mixing chamber; D, capillary tube connecting C and E; E, air chamber; F, sealed end.

(*b*) When sufficient time has elapsed to allow the serum to separate from the clot, break off the ends of the pipette and expel the serum into a sterile watch glass, previously labelled "serum."

(*c*) By means of a pipette* dilute one measure (say 5 cmm.) of the serum with nineteen measures

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* Any simple form of pipette may be used. Wright's are easily made from a piece of glass tubing in the following manner:—One end of the tube is sealed, and at about an inch and a half from this end it is drawn out to form a capillary tube about two inches long and open at its lower end. The thicker part of the tube is again heated at about an inch from its sealed extremity and drawn out so that one finally has a pipette consisting of a couple of bulbs, the upper one sealed at one end and at the other end communicating by a narrow tube with the lower, which in turn communicates with the outer air by the capillary tube. On warming the upper bulb the contained air is rarefied, and as it again cools the partial vacuum which is formed will suck up fluid into the lower bulb. Instead of careful graduation a single mark is made by means of a speck of sealing-wax at a selected point of the capillary tube. Each time the fluid *which is being* aspirated reaches this mark the end of *the tube is raised* for an instant to admit a tiny bubble of air.

(say 95 cmm.) of the saline solution, and expel this into another watch glass marked 1 in 20.

(*d*) With the platinum needle take four loopfuls of the 1 in 20 serum and deposit them separately on the slide or coverglass. Then sterilise the loop, and, when it has cooled, deposit six loopfuls of the bouillon in the same manner. Mix the drops as quickly as possible, place the coverslip on the slide, and seal the edges with vaseline to prevent evaporation. This preparation should then be marked "1 in 50." In cases of difficulty, dilutions of 1 in 30 (four loopfuls of the 1 in 20 serum and two of bouillon) and 1 in 100 (two loopfuls of 1 in 20 serum and eight of bouillon) should also be made, whilst a control experiment must be simultaneously performed by preparing a dilution of three parts of bouillon with two of normal saline solution, and observing that in it the bacilli remain motile and do not tend to form clumps. The slides should then be systematically examined with a magnification of about 300 to 400 diameters. If the serum be taken from a patient who is not the subject of typhoid fever, the bacilli preserve their motility unimpaired, and continue to do so for days if the specimen be suitably preserved. Moreover, they are diffused with tolerable uniformity through the fluid, and show no distinct tendency to form clumps.

If, however, the serum be taken from a patient after the first week of an attack of typhoid fever, or even on the third or fourth day of the illness, though

When this in turn reaches the mark the bulb is again raised, and the process repeated until the required number of measures have been drawn into the lower bulb.

In performing Widal's test extreme accuracy in the degree of dilution is not essential, and therefore ungraduated tubes, such as Wright's, used with reasonable care, meet the requirements of the case. Those who for any special purpose desire a very exact dilution should employ the leucocyte pipette of a Thoma-Zeiss hæmocytometer, or Hawksley's graduated pipette with a screw compressor.

in this case the phenomena will be less distinct, the following facts will be observed:—The motility of the bacilli is almost at once impaired, and after the lapse of a short time their movements wholly cease. They also tend to become agglomerated into clumps, which in a typical case will begin to form soon after the microscopic examination has been commenced, and by the end of half an hour or an hour hardly any solitary bacilli will be found in the specimen. Even where the serum is weaker, as occurs in a certain proportion of cases of typhoid, the same changes eventually supervene, but they occur much more slowly. As a rule, however, impaired motility and a tendency to form clumps are distinctly visible within half an hour, and there are but few cases in which the observations need to be extended over two hours.

In conducting the above examination the following points must be attended to:—(1) The slides, watch glasses, and other pieces of apparatus must be scrupulously clean. If they are not so, appearances of agglutination may occur even where the serum is taken from a man in good health. (2) As one is dealing with bacilli which are alive and virulent, any piece of apparatus which has been used must at once be thoroughly disinfected by heat or by 1 : 500 corrosive sublimate solution, and on no account laid aside before this has been done. (3) Never allow concentrated serum to come into contact with the bouillon culture even for an instant; therefore dilute the serum before mixing it with the bouillon. Concentrated serum from a perfectly healthy person may induce clumping. (4) Let the serum used for examination be free from clot. The presence of red blood corpuscles is comparatively unimportant, but as they can easily be got rid of by centrifuging, it is better to do so.

Widal's reaction may be obtained by employing dead cultures instead of a fresh one in bouillon. The bacilli are killed by heating the bouillon to 60° C. for one hour, or by adding one drop of formaline to every 150 drops of bouillon. This is then kept for use in sealed glass tubes, and has the advantage that it can be utilised when the observer has no laboratory at hand for the cultivation of living bacteria. The agglutination test can also be performed macroscopically; the technique for this method is fully described in a valuable paper by Professor Wright.* Some workers of experience lay stress on the use of more concentrated serum than has been recommended in this account of Widal's reaction, and consider that a complete absence of clumping at the end of fifteen minutes, when a 1 in 10 dilution has been employed, affords a very strong presumption against the serum having been obtained from a patient with typhoid fever.

The agglutination test may be applied to numerous other bacteria, and will be referred to when they are described. The technique in these other cases is identical with that of Widal's test for typhoid.

It need hardly be remarked that to examine a specimen of the stools directly under the microscope, with the view of detecting Eberth's bacillus, is certain to fail in its aim.

5. **Bacillus coli** is a flagellate bacillus which appears under a considerable variety of forms. It is important as the cause of some cases of cystitis and of intra-abdominal suppuration. Some of its forms resemble that of Eberth's bacillus, but can be distinguished by the fact that it produces fermentation in lactose, in which process the medium becomes acid, as can be shown by the addition of litmus, and under suitable conditions gas bubbles are found. To isolate it from other organisms in cases of cystitis, cultures may advantageously be made on MacConkey's taurocholate of soda medium.† The bacillus

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* *Brit. Med. Journ.*, 1898, i., p. 355.

† *See* a paper on "Bile Salt Broth," by MacConkey and *Hill, in the* Thompson Yates Laboratory Reports (1901), *vol. iv., part* i., p. 151.

coli exhibits the agglutinative reaction, but it has been found that serum which agglutinates one species of coli bacillus may fail to produce the reaction in another.

6. **Bacillus choleræ** (Koch) is a curved vibrio with a flagellum at each end. It possesses very marked motility. The organism is much smaller than the bacillus of tuberculosis. Involution forms are rather common. It is readily coloured by basic aniline dyes, but is not stained by Gram's method. It responds to the agglutination test. (FRONTISPIECE, *c.*)

The gelatin cultures, and the so-called cholera-red reaction in broth,* are very characteristic, and should be practised in case of doubt.

In examining the stools of a suspected case, the organism will be found most abundantly present in the mucoid masses. A film should be made from one of these, and, after staining with dilute Ziehl's fluid, examined with an oil immersion lens. In cases where they are fairly numerous, and where their disposition is not too much interfered with by the simultaneous existence of other organisms, they will be seen to lie in rows, end to end, and all pointing in the same direction like fish in a stream.

Equally characteristic is the appearance when a drop of the suspected stool is added to 2 cc. of a decidedly alkaline solution of 1 part of sodium chloride and 10 parts of peptone in 100 parts of water. After eight to twelve hours' incubation at a temperature of 37° C., an abundant growth of the cholera bacillus will be found on the surface of the fluid, and from it plate cultures may be made.

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* The red reaction is due to a nitrosoindol body. Many bacteria produce indol in bouillon; only two, the bacillus of cholera and the bacillus of Finkler, produce both indol and nitrites. The bacillus of cholera produces these substances in the course of a few hours in sufficient amount to give a distinct reaction, Finkler's bacillus only after three or four days' incubation. The addition of an acid capable of acting on the nitrites, and so liberating nitrous acid, is all that is necessary to produce the red coloration; and the best acid to use is pure hydrochloric, as it is less often contaminated with traces of nitrous acid than are nitric and many samples of sulphuric acid, which will frequently produce *the reaction* not only with Finkler's and the cholera *bacillus, but also with* all other bacteria which produce indol.

7. **Bacillus anthracis.**—This organism affects certain classes of persons, especially wool sorters. Its appearance varies considerably with the medium in which it is flourishing. When found in the blood it consists of straight rods fully as long as a red blood corpuscle; often two or more are arranged end to end, either in a straight line or slightly inclined to each other. It stains readily and is coloured by Gram's method.

8. **Bacillus pestis.**—This bacillus is the cause of plague. It commonly gains access through a breach in the skin or mucous membranes, and makes its way by the lymphatic system to the neighbouring glands. In them it leads to the formation of buboes, and if not arrested there passes on into the blood-stream, inducing a septicæmic condition. It is most readily demonstrated in the pus which forms in the buboes, but when septicæmia has developed, bacilli can also be demonstrated in the blood. The appearances presented by the bacillus, alike in cultures and in material derived from patients, are very variable. Its most typical form is that of a small rod with rounded ends, devoid of motility, and probably possessed of a gelatinous capsule. It often presents a characteristic bipolar staining owing to the central segment remaining uncoloured, or only feebly tinged by the dye; this is well brought out when methylene blue is employed, after preliminary treatment with dilute acetic acid. This property is of diagnostic importance, as it is the only bacillus pathogenic in man which is known to possess it. It is decolorised by Gram's method. According to some

authorities it reacts to the agglutination test, the serum of a plague patient becoming efficient in the course of the second week from the incidence of the disease, and remaining so until the eighth week or longer.*

In the report of the Indian Plague Commission of 1898-99,† however, cogent reasons are adduced for questioning the reality of the agglutinative reaction. Their general conclusions on the bacteriological diagnosis of plague may be summarised as follows:—

(1.) Microscopical examination of suspected materials.

(*a*) Pus. Positive or negative results generally alike conclusive. In very early or very late stages negative results may be unreliable.

(*b*) Blood. Positive results generally conclusive. Negative results, unreliable.

(*c*) Sputum. Positive results, reliable if the organisms are fairly characteristic, and if pneumococci are absent.

(*d*) Other materials obtained from surroundings of patient. Results absolutely unreliable, as bacilli morphologically indistinguishable from B. pestis occur outside the body.

(2.) Culture methods.

None can be depended upon to reveal the presence of plague bacilli, when these are mixed with large numbers of contaminating micro-organisms.

(3.) Inoculation methods.

The most suitable animals are guinea-pigs and mice. The death of the inoculated animal only confirms the diagnosis when the B. pestis can be demonstrated in its blood or spleen.

(4.) Serum diagnosis.

"No practical value attaches to the method."

9. **Tetanus, glanders, relapsing fever,** and numerous other rarer diseases are also due to bacilli, but cannot be further referred to here.

II.—MICROCOCCI.

1. **Staphylococci.**—These occur in small masses and are readily stained by ordinary aniline dyes and

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* *Cf.* Deutsche Pest-Kommission, s. 321–324.

† Vol. v., p. 54 *et seq.*

by Gram's method. The individual cocci measure about 1 μ in diameter. The chief varieties are S. pyogenes aureus, S. pyogenes albus, S. citreus.

2. **Streptococci.**—This group contains a considerable number of pathogenic organisms. They occur in shorter or longer chains.

(*a*) **S. pyogenes.**—Stains as the staphylococci. It produces a severe suppuration with a great tendency to indeterminate extension.

(*b*) **S. erysipelatosus** is very closely related to the last, and certainly cannot be distinguished by cover-glass preparations or by culture methods. The chains contain from five or ten to as many as forty cocci. Many authorities regard it as only a virulent form of S. pyogenes.

3. **Pneumococcus** (diplococcus pneumoniæ, Frænkel).—These diplococci are the cause of croupous pneumonia, though they occur also in normal saliva. They are lancet-shaped and the points of the lancets are directed away from each other. The diplococcus is enclosed in a capsule which stains less deeply than the cocci themselves. They stain readily, and are coloured by Gram's method, which aids in distinguishing them from Friedländer's pneumobacillus, as the latter cannot be stained by it. (FRONTISPIECE, *d.*)

4. **Diplococcus intracellularis** (Weichselbaum).—This organism is found within the pus cells of many cases of epidemic cerebro-spinal meningitis, and may be recognised in the fluid removed by lumbar puncture during the life of the patient. In cover-slip preparations the diplococcus can be stained by several aniline dyes; Löffler's methylene blue process yields excellent results. It is decolorised by Gram's method. The cocci are arranged in pairs, the long axes of the individual cocci lying parallel to each other; the diplococcus thus presents a somewhat

striking likeness to the gonococcus. Cultures should be made, in doubtful cases, on glycerine agar. Dr. Still has described a somewhat similar, but less virulent, diplococcus as occurring in the non-tuberculous posterior basic meningitis of children. Still's organism is more easily grown than the true diplococcus intracellularis.

5. **Micrococcus tetragenus.**—This organism is found in the sputum which comes from phthisical, bronchiectatic and other cavities in the lungs. The cocci are about 1·5 μ in diameter, and occur in groups of four enclosed in a capsule. They are readily coloured by Gram's method and by basic aniline dyes.

6. **Gonococcus.**—This is a diplococcus, the two cocci of which lie very close together and are thus difficult to distinguish. With high magnification they are seen to be kidney-shaped and have their concave sides facing one another. Sometimes the gonococci are found free; at other times, and more characteristically, enclosed in pus corpuscles, in which they occur in groups. They are readily stained by aqueous solutions of basic aniline dyes. They are decolorised by Gram's method, which often aids in clinching the diagnosis in a doubtful case. Advantage may also be taken of the fact to secure a double stain, the cover glass being first treated by Gram's method and then counterstained with vesuvin or Bismarck brown. Most of the other microbes then stain violet, the cellular elements are light brown, and gonococci are darker brown. (FRONTISPIECE, *e.*)

To determine the identity of the gonococcus in doubtful cases the following criteria are available* :—

i. The occurrence in the pus cells of paired bean-shaped cocci. At least four pairs in each cell should be recognised.

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* Curtis, "Essentials of Practical Bacteriology," p. 127 *(After Foulerton.)*

ii. The typical cocci should be readily decolorised when treated by Gram's method.

iii. Pure growths cannot be subcultured on gelatin at 20° C. or on agar at either 20° C. or 36° C.

7. **Micrococcus Melitensis** (Bruce) is a very minute oval coccus about one-third of a micromillimetre in diameter. In coverslip preparations it is readily stained by basic aniline dyes, but it is decolorised by Gram's method. The cocci are for the most part arranged singly, but sometimes they form short chains. The micrococcus Melitensis exhibits the agglutinative reaction very distinctly, and in doubtful cases the diagnosis may, in this manner, be established. The blood serum from the suspected patient should be mixed with a pure culture in exactly the same manner as has been detailed for typhoid fever. By this means Wright and Smith* have been able to prove that some of the Indian fevers are due to the same organism as Malta fever.

8. **Sarcina ventriculi** is described elsewhere (p. 91).

III.—ACTINOMYCES.

The ray fungus (Figs. 85 and 147) has been placed in various groups of fungi by different observers. It has affinities with several classes, but is perhaps best associated in the meantime with a somewhat miscellaneous group under the heading "Fungi imperfecti," as has been proposed by Winter.† It may be found in pus, sputum, fæces, urine, or in tissues removed by operation or after death. It occurs in the form of minute yellow granular masses, which are generally recognised without much difficulty under the microscope. The most expeditious method for its demonstration is to crush a small granule, immersed in a drop of glycerine, between a microscopic slide and cover glass. Treated thus, one sees a radiating cluster of pear-shaped bodies, whilst in the centre of the mass the filaments of an interlaced mycelium may be observed. If a stained preparation is wanted, a good method* is to desiccate a layer of pus on a cover glass, then to wash it with ether and immerse in a concentrated solution of caustic potash. From this it is transferred to a 5 per cent. solution of eosin in water, where it remains for a quarter of an hour. It is finally washed in a concentrated solution of acetate of potash and mounted in the same medium. The centre of the mass is stained a bright red, the clubs pale pink or yellow.

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* *Lancet*, 1897, i., p. 656.

† *Rabenhorst.* Kryptogamenflora. Bd. I. Die Pilze, bearb. *von Dr.* Georg Winter. The present tendency is to regard it *as more* closely related to the streptothrix section of bacteria *than to* any other group.

The mycelial elements are best stained by Gram's method; thionine blue also yields satisfactory preparations.

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* Lemiere and Bécue.

APPENDIX.

WEIGHTS AND MEASURES

1. English weights and measures.

1 grain, gr.
1 ounce, oz. = 437·5 grains.
1 pound, lb. = 16 ounces = 7,000 grains
1 minim = 0·91146 grain.
1 fluid drachm = 60 minims.
1 fluid ounce = 8 fluid drachms
1 pint = 20 fluid ounces
1 gallon = 8 pints.

2 Relation of English to metric system.

1 grain = 64·8 milligrammes.
1 ounce = 28·3 grammes.
1 lb. = 453·6 grammes
1 gramme = 15·432 grains.
1 kilo = 2 lb. 3 oz.
1 minim = 0·059 cc.
1 fluid drachm = 3·5 cc.
1 fluid ounce = 28·39 cc.
1 pint = 567·9 cc.
1 cc. = 16·9 minims.
1 litre = 35·2 fluid ounces.
1 inch = 2·54 cm.
1 foot = 30·48 cm.
1 yard = 91·44 cm.
1 cm. = 0·39 in.
1 metre = 39·37 in.

3. Conversions.

To convert grammes per 100 cc. into grains pe *ounce*, multiply by 4·375.

To convert grammes into ounces avoirdupois multiply by 10 and divide by 283.

To convert litres into pints, multiply by 88 and divide by 50.

To convert kilos into pounds, multiply by 1,000 and divide by 454.

4. Centigrade and Fahrenheit scales.

To convert Fahrenheit into Centigrade, subtract 32, multiply the remainder by 5, and divide the result by 9.

To convert Centigrade into Fahrenheit, multiply by 9, divide by 5, and add 32.

The following table and figure show the relation of degrees Fahrenheit to Centigrade, as far as is likely to be required in clinical work :—

Centigrade.		Fahrenheit.	Centigrade.		Fahrenheit.
110	...	230	37	...	98·6
100	...	212	36·5	...	97·7
95	...	203	36	...	96·8
90	...	194	35·5	...	95·9
85	...	185	35	...	95·0
80	...	176	34	...	93·2
75	...	167	33	...	91·4
70	...	158	32	...	89·6
65	...	149	31	...	87·8
60	...	140	30	...	86
55	...	131	25	...	77
50	...	122	20	...	68
45	...	113	15	...	59
44	...	111·2	10	...	50
43	...	109·4	+ 5	...	41
42	...	107·6	0	...	32
41	...	105·8	− 5	...	23
40·5	...	104·9	− 10	...	14
40	...	104·0	− 15	...	+ 5
39·5	...	103·1	− 20	...	− 4
39	...	102·2	0·54°	=	1°
38·5	...	101·3	1	=	1·8
38	...	100·4	2	=	3·6
37·5	...	*99·5*	2·5	=	4·5

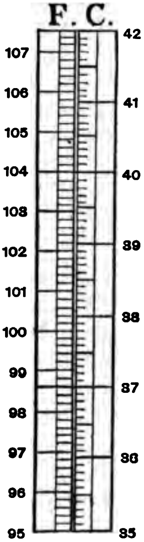

Fig. 150. Fahrenheit and Centigrade scales compared.

SOLUTIONS REQUIRED FOR EXAMINATION OF GASTRIC CONTENTS.

5. Phloroglucin and vanillin solution.

Dissolve 2 grms. phloroglucin and 1 grm. of vanillin in 30 cc. of absolute alcohol. Keep the solution in the dark, and use it economically.

6. Boas's resorcin reagent.

Resorcin	75 grs.
White sugar	45 grs.
Dilute spirit	3½ oz.

Dissolve.

7. Uffelmann's reagent.

Carbolic acid (1 in 20) ...	10 cc.
Distilled water	20 cc.

Mix.

Add one or two drops of liq. ferri perchlor. An amethyst blue solution results. It should be prepared fresh each time, as it does not keep. Lactic acid turns it yellow. Hydrochloric acid simply discharges the blue colour. Acetic and *combined* hydrochloric acid turn it somewhat brownish.

8. Congo red test papers.

These are made by soaking bibulous paper in a solution of Congo red, of the strength of 1 decigramme to 100 cc. of water, or in a saturated alcoholic solution. They are allowed to dry, and are then ready for use.

SOLUTIONS REQUIRED FOR URINARY TESTING.

9 Standard nitrate of silver solution.

Dissolve 29·063 grms. of pure *fused* silver nitrate in distilled water, and fill up to 1 litre. Keep in the dark.

10. **Standard uranium solution.**

Dissolve 3·5 grms. of uranium nitrate in 90 cc. of water to which has been added 2·5 cc. of glacial acetic acid; then fill up to 100 cc.

$$1 \text{ cc.} = 5 \text{ mg. } P_2O_5.$$

11. **Acetic solution of sodium acetate.**

Dissolve 100 grms. of crystals of sodium acetate in some water; add 100 cc. of strong acetic acid, and dilute with water to 1 litre.

12. **Hypobromite solution.**

Dissolve 100 grms. of caustic soda in 250 cc. of water. Cool, then add 25 cc. of bromine. The solution is apt to undergo the following decomposition:—

$$3\,NaBrO = 2\,NaBr + NaBrO_3.$$

It is therefore better to prepare it as required by adding 2·5 cc. of bromine to 25 cc. of the caustic soda solution.

The bromine is supplied in small tubes, which readily break when shaken up smartly with the soda solution in a stout stoppered bottle.

13. **Esbach's reagent.**

Dissolve 10 grms. of picric acid and 20 grms. of citric acid in about 900 cc. of boiling water; cool, and add water to 1 litre.

14. **Fehling's solution.**

(*a*) Take 34·64 grms. of pure sulphate of copper which has been powdered and pressed between bibulous paper, dissolve in 200 cc. of warm distilled water, cool, and fill up to 500 cc.

(*b*) Dissolve 180 grms. of crystallised Rochelle salt in 300 cc. of hot water, filter, and add 70 grms. of pure caustic soda, or 100 grms. of potash; cool, fill up to 500 cc.

When required, mix equal volumes of (*a*) and (*b*). The result is an alkaline solution of potassic cupric tartrate, of which 1 cc. is exactly reduced by 5 mg. of pure glucose.

14*a*. **Nylander's (or Böttger's) reagent.**

Dissolve 10 grms. of caustic soda in 100 ccm. of water, warm, and add 4 grms. of sodio-potassium tartrate and 2 grms. of bismuth subnitrate. Shake thoroughly. A hydrated oxide of bismuth is formed, which is kept in solution by the tartrate. The solution should be filtered and kept in a tightly stoppered bottle in the dark.

15. **Pavy's solution.**

Required—

Cupric sulphate	4·158 grms. (36½ grs.).
Rochelle salt	20·4 grms. (178 grs.).
Caustic potash	20 4 grms.
Strong ammonia	300 cc.
(Specific gravity, 0·880)	(6 oz.).
Water to	1 litre. (1 pt.).

Dissolve the Rochelle salt and potash in part of the water, and the sulphate of copper in another (with the aid of heat), pour the copper solution into that of the alkali and Rochelle salt, cool, add the ammonia, then fill up to 1 litre or 1 pt.

Keeps indefinitely. 10 cc. = 5 mg. glucose.

16. **Peptone solution for testing for bile acids.**

Powdered peptone (Savory and Moore's)	½ dr.
Salicylic acid	4 grs.
Acetic acid	½ dr.
Distilled water to	8 oz.

Filter repeatedly until transparent.

SOLUTIONS REQUIRED IN THE EXAMINATION OF BLOOD.

17. **Diluting fluid for hæmocytometer.**

Sulphate of soda	104 grs.
Acetic acid	1 dr.
Distilled water	6 oz.

18. **Hayem's solution.**

Common salt	1 grm.
Sulphate of soda	5 grms.
Corrosive sublimate	0·5 grm.
Distilled water	200 cc.

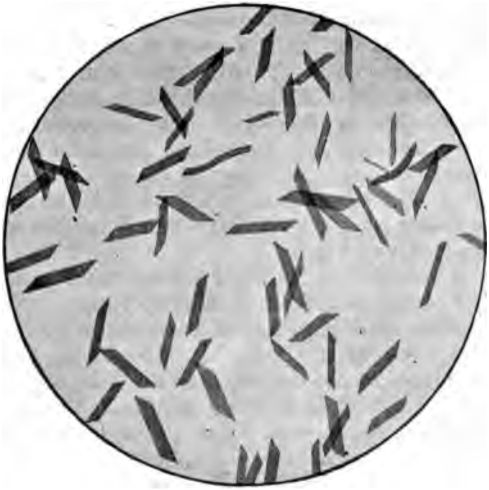

Fig. 151.—Hæmin Crystals. × 830.

19. **Teichmann's test for blood (hæmin test).**

Take up in a pipette some of the deposit to be examined. Rub it up with a small amount of common salt, and evaporate a little of the mixture to dryness on a slide. Moisten the residue with glacial acetic acid, and put on a cover glass. Gently heat this over a very small flame for several minutes, avoiding boiling. Allow a little glacial acetic acid to run in from the side of the cover glass from time to time during the process. Allow to cool, and examine for hæmin crystals with a high power (Fig. 151).

SOME STAINING METHODS.

20. **Gram's method.**

The following solutions are required :—

(1) A solution of gentian violet in aniline water.

This is prepared as follows :—

Place in a test tube 1 part of aniline oil and 20 of ordinary water. Shake these thoroughly together, and filter. Preserve the filtrate (*a*) in a stoppered bottle in the dark. Prepare a saturated alcoholic solution of gentian violet and filter it (*b*). To 9 parts of (*a*) add 1 part of (*b*) and filter the mixture. The two solutions should only be mixed shortly before use; after twenty-four hours the stain becomes less trustworthy.

(2) A solution of 1 grm. of iodine and 2 grms. of iodide of potash in 300 cc. of distilled water.

Float the cover slip, face downwards, in a capsule containing some of the stain. In the cold the film is usually stained in five minutes; if the fluid is heated till steam rises, in about one minute. Wash the cover slip in water, then place it for half to one minute in solution 2. Here the film becomes black. It is then washed in alcohol or methylated spirit until it ceases to lose colour and becomes a pale grey. Wash in water. Dry between filter papers. Mount in xylol balsam.

The following modification of Gram's method will be found simpler and more efficient. For aniline oil water substitute a 1 in 20 aqueous solution of phenol in the same proportions, and carry out the staining as detailed above. After treatment with the iodine solution, and washing with alcohol, transfer to clove oil, which increases the sharpness of the differentiation; wash out the clove oil with alcohol, then transfer to water, employ a contrast stain if desired, wash again in water, dry, and mount in xylol balsam.

Some bacteria retain this stain, especially tubercle, leprosy, diphtheria, tetanus, yellow fever, and anthrax amongst bacilli; and streptococci, staphylococci, micrococcus tetragenus, and diplococcus pneumoniæ (Frænkel) amongst cocci; but many are decolorised by the process, and are thereby distinguished from those already mentioned, the most important being plague, cholera, influenza, glanders, typhoid, bacillus coli communis, diplococcus intracellularis, gonococcus, and micrococcus Melitensis.

21. **Ziehl-Neelsen stain.**

A. Fuchsin, 1 part
Absolute alcohol, 10 parts } dissolve.

Add of 5 per cent. aqueous solution of phenol 100 parts.

B. Twenty per cent. sulphuric acid.

C. Watery solution of methylene blue. The solution should be nearly saturated. The addition of a trace of ammonia increases the precision of the staining.

Method.—Heat A till steam rises, then float cover glasses, film down, on it for three or four minutes, rinse in water, immerse in B till decolorised, wash in water, counterstain if desired in C for one minute or more, wash rapidly in water, dry, and mount in xylol balsam for permanent preparations, or Farrant's medium if not to be preserved. Sections *require longer* staining and must not be dried ; clove oil should not be used for clearing purposes, as it often decolorises the bacteria.

22. **Löffler's stain.** (This should be freshly prepared.)

Concentrated alcoholic solution of methylene blue	1 cc.
Caustic potash in 00·1 per cent. aqueous solution	3 cc.

Specimens are stained in from five to thirty minutes. Excess of stain is discharged by rapid washing in water acidulated with acetic acid (2 drops of acid in a watch-glassful of water) and all traces of acid well washed out. The specimen is then dried and mounted.

23. **Carbol thionine.** (Prepared freshly.)

Saturated solution of thionine in 50 per cent. alcohol	10 cc.
1 in 40 solution of phenol in water	100 cc.

This stain is one of the best for film preparations. After staining, which is rapidly effected, wash the specimen in water, then dry and mount. Sections should, after washing, be passed through alcohol containing a trace of ammonia, thereafter dehydrated by absolute alcohol, cleared with xylol, and mounted in balsam.

24. **Aniline water** is made by shaking up 1 part of *colourless* aniline oil with 3 parts of distilled water in a bottle of dark glass. The excess of oil sinks to the bottom, and the supernatant aniline water is decanted and filtered through a filter-paper previously moistened with distilled water, when it is ready for use. Both the aniline oil and the aniline water must be kept in bottles of dark glass.*

* It is now usual to substitute a 1 in 20 aqueous solution of phenol for aniline water. It is more readily prepared, keeps better, and its mordant action is quite as efficient.

25. **Ehrlich's Triacid Stain.**—Prepare saturated watery solutions of *chemically pure* and crystalline orange G, acid fuchsin and methyl green. Then make the following mixture :—

Orange G solution	13–14 cc.
Acid fuchsin solution	6–7 cc.
Distilled water	15 cc.
Alcohol	15 cc.
Methyl green solution	12·5 cc.
Alcohol	10 cc.
Glycerine	10 cc.

The fluids must be measured out in the above order, in the same glass, and from the addition of the methyl green onwards the mixture is thoroughly shaken. The solution can be used at once and keeps indefinitely.

Blood films stain in it in from one to five minutes, depending upon the particular blood under examination and the mode of its fixation. The exact time required can therefore only be found out by experiment.

26. **Van Ermengen** recommends the following solution for cleansing slides and cover glasses :—

Concentrated sulphuric acid	6 parts.
Potassium bichromate	6 ,,
Water	100 ,,

Leave the glasses in the above solution for twelve hours ; wash in water until all trace of bichromate is gone ; thereafter preserve in absolute alcohol.

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Use of Polariscope

1. Know instrument
2. Use clear urine
   Kieselguhr
   PbAc - Sugar of lead
   (90 cc URINE + 10 cc 25% PbAc)
3. Arrange tube carefully.
4. Always see zero point
   make 4 readings &
   take average.
5. Focus for sharp line
   then rotate (1° = 1% sugar.)

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